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09/189415

? b 411
17jul00 18:22:44 User231835 Session D593.2
? sf all
? s (intimin or tir) and ((amino acid)(10n)(sequence?))
? s (intimin or tir) and sequence?

Your SELECT statement is:
s (intimin or tir) and sequence?
? s (intimin or tir)(20n)(sequence?) and coli
s (intimin or tir)(20n)(sequence?) and coli
? s ((intimin or tir)(20n)(sequence?)) and coli
? s ((intimin or tir)(20n)(sequence?)) and coli
S1 336 ((INTIMIN OR TIR)(20N)(SEQUENCE?)) AND COLI
? s s2 and ((94 or 78 or 94000 or 78000)(10n)(kd or kda or kilodalton? or dalton?))
S3 12 S2 AND ((94 OR 78 OR 94000 OR 78000)(10N)(KD OR KDA OR
KILODALTON? OR DALTON?))
? t s3/3,kwic/1-12

3/3,KWIC/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11432013 BIOSIS NO.: 199800213345
Molecular analysis of Shiga toxigenic Escherichia %%%coli%%% O111:H- proteins which react with sera from patients with hemolytic-uremic syndrome.
AUTHOR: Voss Elena; Paton Adrienne W; Manning Paul A; Paton James C(a)
AUTHOR ADDRESS: (a)Mol. Microbiol. Unit, Women's Child. Hosp., North Adelaide, SA 5006**Australia
1998
JOURNAL: Infection and Immunity 66 (4):p1467-1472 April, 1998
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

Molecular analysis of Shiga toxigenic Escherichia %%%coli%%% O111:H- proteins which react with sera from patients with hemolytic-uremic syndrome.

...ABSTRACT: of hemolytic-uremic syndrome (HUS) caused by fermented sausage
contaminated with Shiga toxin-producing Escherichia %%%coli%%% (STEC).
The predominant STEC isolated from HUS patients belonged to serotype O111:H-, and reactivity...

...response, but several protein bands were also immunoreactive, particularly one with an apparent size of %%%94%%% %%%kDa%%%. One
convalescent-phase serum sample was subsequently used to screen an O111:H- cosmid bank...

...blot analysis of one of these clones identified three major immunoreactive protein bands of approximately %%%94%%%, 70, and 50 %%%kDa%%%. An immune response to the three proteins was detectable with
all five convalescent-phase serum samples but not with healthy human serum. Immunoreactive %%%94%%%- and 50-%%%kDa%%% species were produced by
a deletion derivative of the cosmid containing a 7-kb STEC...

...it is part of the locus for enterocyte effacement, including the eaeA gene which encodes %%%intimin%%%. The deduced amino acid %%%sequence%%%
of the O111:H- %%%intimin%%% was 88.6% identical to %%%intimin%%% from
O157:H7 STEC, and the most divergent region was the 200 residues at the ...

...HUS patient infected only with the O111:HSTEC reacted with intimin from an enteropathogenic E. %%%coli%%% O111 strain, as well as several other eaeA-positive STEC isolates, but not with an...
DESCRIPTORS:
...ORGANISMS: Escherichia-%%%coli%%% (Enterobacteriaceae...

3/3,KWIC/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2000 BIOSIS. All rts. reserv.

09825552 BIOSIS NO.: 199598280470
A Plasmid-Encoded Regulatory Region Activates Chromosomal eaeA Expression
in Enteropathogenic Escherichia %%%coli%%%.
AUTHOR: Gomez-Duarte Oscar G; Kaper James B(a)
AUTHOR ADDRESS: (a)Cent. Vaccine Dev., Div. Geographic Med., Univ. Maryland
Sch. Med., 10 S. Pine St., Baltimore, M**USA
1995
JOURNAL: Infection and Immunity 63 (5):p1767-1776 1995
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

A Plasmid-Encoded Regulatory Region Activates Chromosomal eaeA Expression
in Enteropathogenic Escherichia %%%coli%%%.

ABSTRACT: Enteropathogenic Escherichia %%%coli%%% (EPEC) organisms produce
a characteristic histopathology in intestinal epithelial cells called attaching and effacing lesions. The eaeA gene is associated with attaching and effacing lesions and encodes intimin, a
%%%94%%%-%%%kDa%%%
outer membrane protein. A 60-MDa plasmid, pMAR2, is essential for full virulence of EPEC...

...by increased alkaline phosphatase activity of an eaeA::TnphoA gene fusion, increased expression of the %%%intimin%%% protein, and increased production of eaeA mRNA. These %%%sequences%%% are called per for plasmid-encoded regulator. pMAR2-cured JPN15 containing cloned per sequences adheres...
DESCRIPTORS:
ORGANISMS: Escherichia %%%coli%%% (Enterobacteriaceae)

3/3,KWIC/3 (Item 1 from file: 76)
DIALOG(R)File 76:Life Sciences Collection
(c) 2000 Cambridge Sci Abs. All rts. reserv.

02311981 4417324
Molecular analysis of Shiga toxigenic Escherichia %%%coli%%% O111:H super(-) proteins which react with sera from patients with hemolytic-uremic syndrome
Voss, E.; Paton, A.W.; Manning, P.A.; Paton, J.C.
Molecular Microbiology Unit, Women's and Children's Hospital, North Adelaide, S.A., 5006, Australia
INFECT. IMMUN. vol. 66, no. 4, pp. 1467-1472 (1998)
ISSN: 0019-9567
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Microbiology Abstracts B: Bacteriology

Molecular analysis of Shiga toxigenic Escherichia %%%coli%%% O111:H super(-) proteins which react with sera from patients with hemolytic-uremic syndrome

...of hemolytic-uremic syndrome (HUS) caused by fermented sausage contaminated with Shiga toxin-producing Escherichia %%%coli%%% (STEC). The
predominant STEC isolated from HUS patients belonged to serotype O111:H super(-), and...
...response, but several protein bands were also immunoreactive, particularly one with an apparent size of %%%94%%% %%%kDa%%%. One convalescent-phase serum sample was subsequently used to screen an O111:H super(-) cosmid...

...blot analysis of one of these clones identified three major immunoreactive protein bands of approximately %%%94%%%, 70, and 50 %%%kDa%%%. An immune response to the three proteins was detectable with all
five convalescent-phase serum samples but not with healthy human serum. Immunoreactive %%%94%%%- and 50-%%%kDa%%% species were

produced by a
deletion derivative of the cosmid containing a 7-kb STEC...

...it is part of the locus for enterocyte effacement, including the eaeA gene which encodes %%%intimin%%%. The deduced amino acid %%%sequence%%% of
the O111:H super(-) %%%intimin%%% was 88.6% identical to %%%intimin%%% from
O157:H7 STEC, and the most divergent region was the 200 residues at the...

...infected only with the O111:H super(-) STEC reacted with intimin from an enteropathogenic E. %%%coli%%% O111 strain, as well as several other eaeA-positive STEC isolates, but not with an...
DESCRIPTORS: hemolytic uremic syndrome; proteins; immune response; serum;
immunoblotting; Shiga toxin; Escherichia %%%coli%%%

3/3,KWIC/4 (Item 2 from file: 76)
DIALOG(R)File 76:Life Sciences Collection
(c) 2000 Cambridge Sci Abs. All rts. reserv.

01944539 3770487
A plasmid-encoded regulatory region activates chromosomal eaeA expression in enteropathogenic Escherichia %%%coli%%%
Gomez Duarte, O.G.; Kaper, J.B.
Cent. Vaccine Dev., Div. Geogr. Med., Univ. Maryland Sch. Med., 10 South Pine St., Baltimore, MD 21201, USA
INFECT. IMMUN. vol. 63, no. 5, pp. 1767-1776 (1995)
ISSN: 0019-9567
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Microbiology Abstracts B: Bacteriology

A plasmid-encoded regulatory region activates chromosomal eaeA expression in enteropathogenic Escherichia %%%coli%%%

Enteropathogenic Escherichia %%%coli%%% (EPEC) organisms produce a characteristic histopathology in intestinal epithelial cells called attaching and effacing lesions. The eaeA gene is associated with attaching and effacing lesions and encodes intimin, a %%%94%%%-%%%kDa%%% outer
membrane protein. A 60-MDa plasmid, pMAR2, is essential for full virulence of EPEC...
...by increased alkaline phosphatase activity of an eaeA::TnphoA gene fusion, increased expression of the %%%intimin%%% protein, and increased production of eaeA mRNA. These %%%sequences%%% are called per for plasmid-encoded regulator. pMAR2-cured JPN15 containing cloned per sequences adheres...
DESCRIPTORS: Escherichia %%%coli%%%; eaeA gene; chromosomes; plasmids

3/3,KWIC/5 (Item 1 from file: 349)
DIALOG(R)File 349:PCT Fulltext
(c) 2000 WIPO/MicroPatent. All rts. reserv.

00679850
MEMBRANE-BOUND PROTEINS AND NUCLEIC ACIDS ENCODING THE SAME
PROTEINES MEMBRANAIRES ET ACIDES NUCLEIQUES CODANT CES PROTEINES
Patent Applicant/Assignee:
GENENTECH INC; Address - GENENTECH, INC. , 1 DNA Way, South San
Francisco, CA 94080-4990 , US
Inventor(s):
BAKER Kevin; Address - BAKER, Kevin , 14006 Indian Run Drive, Damestown,
MD 20878 , US
CHEN Jian; Address - CHEN, Jian , 22-03 Hunters Glen Drive, Plainsboro,
NJ 08536-3854 , US
GODDARD Audrey; Address - GODDARD, Audrey , 110 Congo Street, San
Francisco, CA 94131 , US
GURNEY Austin L; Address - GURNEY, Austin, L. , 1 Debbie Lane, Belmont,
CA 94002 , US
SMITH Victoria; Address - SMITH, Victoria , 19 Dwight Road, Burlingame,
CA 94010 , US
WATANABE Colin K; Address - WATANABE, Colin, K. , 128 Corliss Drive,
Moraga, CA 94556 , US
WOOD William I; Address - WOOD, William, I. , 35 Southdown Court,
Hillsborough, CA 94010 , US
YUAN Jean; Address - YUAN, Jean , 176 West 37th Avenue, San Mateo, CA
94403 , US
Patent and Priority Information (Country, Number, Date):
Patent: WO 9963088 A2 19991209
Application: WO 99US12252 19990602 (PCT/WO US9912252)

3/3,KWIC/6 (Item 2 from file: 349)
DIALOG(R)File 349:PCT Fulltext
(c) 2000 WIPO/MicroPatent. All rts. reserv.

00661790
METHODS FOR ASSAYING TYPE III SECRETION INHIBITORS
PROCEDES D'ANALYSE D'INHIBITEURS DE SECRETION DE TYPE III
Patent Applicant/Assignee:
UNIVERSITY OF BRITISH COLUMBIA; Address - UNIVERSITY OF BRITISH COLUMBIA
, I.R.C. Building, Room 331, 2194 Health Sciences Mall, Vancouver, British Columbia V6T 1Z3 , CA
Inventor(s):
FINLAY Brett B; Address - FINLAY, Brett, B. , Biotechnology Laboratory, 237-6174 University Boulevard, Vancouver, British Columbia V6T 1Z3 , CA
KENNY Brendan; Address - KENNY, Brendan , First floor flat, 59 Manor Park, Redland, Bristol BS6 7HW , GB
STEIN Marcus; Address - STEIN, Marcus , Via Fiorentina, II, I-53100 Siena , IT
Patent and Priority Information (Country, Number, Date):
Patent: WO 9945136 A1 19990910
Application: WO 99CA183 19990305 (PCT/WO CA9900183)
Priority Application: US 9876980 19980305
Designated States: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU;
CZ; DE; DK; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP;
KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX;
NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG;
UZ; VN; YU; ZW; GH; GM; KE; LS; MW; SD; SL; SZ; UG; ZW; AM; AZ; BY; KG;
KZ; MD; RU; TJ; TM; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT;
LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN;
TD; TG
Publication Language: English
Filing Language: English
Fulltext Word Count: 14059

3/3,KWIC/7 (Item 3 from file: 349)
DIALOG(R)File 349:PCT Fulltext
(c) 2000 WIPO/MicroPatent. All rts. reserv.

00657883
METHODS
PROCEDES
Patent Applicant/Assignee:
IMPERIAL COLLEGE OF SCIENCE TECHNOLOGY AND MEDICINE; Address - IMPERIAL
COLLEGE OF SCIENCE, TECHNOLOGY AND MEDICINE , London SW7 2AZ , GB
Inventor(s):
BATCHELOR Miranda; Address - BATCHELOR, Miranda , Imperial College of
Science, Technology and Medicine, Dept. of Biochemistry, London SW7 2AZ
, GB
DOUGAN Gordon; Address - DOUGAN, Gordon , Imperial College of Science,
Technology and Medicine, Dept. of Biochemistry, London SW7 2AZ , GB
FRANKEL Gad; Address - FRANKEL, Gad , Imperial College of Science,

Technology and Medicine, Dept. of Biochemistry, London SW7 2AZ , GB
Patent and Priority Information (Country, Number, Date):
Patent: WO 9941614 A2 19990819
Application: WO 99GB467 19990216 (PCT/WO GB9900467)
Priority Application: GB 983322 19980216
Designated States: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU;
CZ; DE; DK; EE; ES; FI; GB; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE;
KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO;
NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; US;
UZ; VN; YU; ZW; GH; GM; KE; LS; MW; SD; SZ; UG; ZW; AM; AZ; BY; KG; KZ;
MD; RU; TJ; TM; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU;
MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD;
TG
Publication Language: English
Filing Language: English
Fulltext Word Count: 11071

3/3,KWIC/8 (Item 4 from file: 349)
DIALOG(R)File 349:PCT Fulltext
(c) 2000 WIPO/MicroPatent. All rts. reserv.

00641460
HP90: HOST MEMBRANE RECEPTOR FOR PATHOGENIC BACTERIA, ENCODED BY THE
BACTERIAL TIR GENE
HP90: RECEPTEUR HOTE A MEMBRANE POUR BACTERIES PATHOGENES CODEES PAR LE
GENE BACTERIEN TIR
Patent Applicant/Assignee:
UNIVERSITY OF BRITISH COLUMBIA; Address - UNIVERSITY OF BRITISH COLUMBIA
, 2222 Health Sciences Mall, Vancouver, British Columbia V6T 1Z3 , CA
Inventor(s):
FINLAY B Brett; Address - FINLAY, B., Brett , Biotechnology Laboratory,
237-6174 University Boulevard, Vancouver, British Columbia V6T 1Z3 , CA
KENNY Brendan; Address - KENNY, Brendan , Biotechnology Laboratory,
237-6174 University Boulevard, Vancouver, British Columbia V6T 1Z3 , CA
DEVINNEY Rebekah; Address - DEVINNEY, Rebekah , Biotechnology Laboratory,
237-6174 University Boulevard, Vancouver, British Columbia V6T 1Z3 , CA
STEIN Marcus; Address - STEIN, Marcus , Biotechnology Laboratory,
237-6174 University Boulevard, Vancouver, British Columbia V6T 1Z3 , CA
Patent and Priority Information (Country, Number, Date):
Patent: WO 9924576 A1 19990520
Application: WO 98CA1042 19981110 (PCT/WO CA9801042)
Priority Application: US 9765130 19971112
Designated States: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU;
CZ; DE; DK; EE; ES; FI; GB; GE; GH; GM; HR; HU; ID; IL; IS; JP; KE; KG;
KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ;
PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; UZ; VN;
YU; ZW; GH; GM; KE; LS; MW; SD; SZ; UG; ZW; AM; AZ; BY; KG; KZ; MD; RU;
TJ; TM; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL;
PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
Publication Language: English
Filing Language: English
Fulltext Word Count: 23409

Fulltext Availability:
Detailed Description
Claims

3/3,KWIC/9 (Item 5 from file: 349)
DIALOG(R)File 349:PCT Fulltext
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00538751
METHOD OF STIMULATING AN IMMUNE RESPONSE BY ADMINISTRATION OF HOST
ORGANISMS THAT EXPRESS INTIMIN ALONE OR AS A FUSION PROTEIN WITH ONE OR
MORE OTHER ANTIGENS
PROCEDE DE STIMULATION D'UNE REACTION IMMUNITAIRE PAR ADMINISTRATION
D'ORGANISMES HOTES QUI EXPRIMENT L'INTIMINE SEULE OU SOUS FORME DE
PROTEINE DE FUSION ASSOCIEE A UN OU PLUSIEURS ANTIGENES
Patent Applicant/Assignee:
HENRY M JACKSON FOUNDATION FOR THE ADVANCEMENT OF MILITARY MEDICINE;
Address - HENRY M. JACKSON FOUNDATION FOR THE ADVANCEMENT OF MILITARY
MEDICINE , Suite 600, 1401 Rockville Pike, Rockville, MD 20852 , US
Inventor(s):
STEWART Charles N; Address - STEWART, Charles, N. , 2105 Rolling Road,
Greensboro, NC 27403 , US
MCKEE Marian L; Address - MCKEE, Marian, L. , 929 Holly Creek Drive,
Great Falls, VA 22066 , US
O'BRIEN Alison D; Address - O'BRIEN, Alison, D. , 5514 Charlotte Road,
Bethesda, MD 20817 , US
WACHTEL Marian R; Address - WACHTEL, Marian, R. , 18705 Walkers Choice
Road &2, Gaithersburg, MD 20879 , US
Patent and Priority Information (Country, Number, Date):
Patent: WO 9740177 A1 19971030
Application: WO 97US5831 19970418 (PCT/WO US9705831)
Priority Application: US 9615657 19960419; US 9615938 19960422
Designated States: AU; CA; JP; AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE;
IT; LU; MC; NL; PT; SE
Publication Language: English
Filing Language: English
Fulltext Word Count: 25314

Fulltext Availability:
Detailed Description

English Abstract
This invention satisfies needs in the art by providing intimin, the Enterohemorrhagic < i> Escherichia %%%coli%%% < /i> (EHEC) adherence protein, alone or as a fusion protein with one or more other...

French Abstract
...invention se rapporte a l'administration d'intimine, la proteine d'adherence a < i> Escherichia %%%coli%%% < /i> enterohemorragique (EHEC), seule ou sous forme de proteine de fusion associee a un ou...

Detailed Discription
... OF THE INVENTION
A virulent form of bloody diarrhea is caused by the Enterohemorrhagic Escherichia %%%coli%%% (EHEC). This pathogen is the most common infectious cause of bloody diarrhea (also called hemorrhagic...1989)).

In 1990, Jerse et aL identified a chromosomal gene in a related diarrheagenic E %%%coli%%% strain, Enteropathogenic E %%%coli%%% (EPEC).
That gene, designated eae, was found to be required for the bacterium to produce...

Purification of the protein is accomplished...fusion proteins remain primarily in the insoluble pellet after sonic disruption of the host E. %%%coli%%%. Therefore, urea and guanidine HCl are included in the purification protocol, which allows extraction of...

...with whether the protein is expressed alone and whether cross-immunity is desired with an %%%intimin%%%-like protein of known amino acid %%%sequence%%%. In addition to %%%intimin%%% expressed from EHEC and

EPEC, examples of %%%intimin%%%-like proteins include, but are not limited to, %%%intimin%%%-like proteins of Citrobacter rodentium, Hafina alveii, and the invasins of Yersinia enterocolitica and Yersinia heightened desire for cross-immunity with Enteropathogenic Escherichia %%%coli%%% (EPEC), Citobacter rodentium, or Hafnia alvei. EPEC intimin
shares 83% identity with EHEC intimin over...

3/3,KWIC/10 (Item 6 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00538738
HISTIDINETAGGED INTIMIN AND METHODS OF USING INTIMIN TO STIMULATE AN
IMMUNE RESPONSE AND AS AN ANTIGEN CARRIER WITH TARGETING CAPABILITY
INTIMINE MARQUEE A L'HISTIDINE ET PROCEDES D'UTILISATION DE L'INTIMINE POUR
STIMULER UNE REACTION IMMUNITAIRE ET EN TANT QUE PORTEUR D'ANTIGENE A
CAPACITE DE CIBLAGE
Patent Applicant/Assignee:
HENRY M JACKSON FOUNDATION FOR THE ADVANCEMENT OF MILITARY MEDECINE;
Address - HENRY M. JACKSON FOUNDATION FOR THE ADVANCEMENT OF MILITARY
MEDECINE , Suite 600, 1401 Rockville Pike, Rockville, MD 20852 , US
Inventor(s):
MCKEE Marian L; Address - MCKEE, Marian, L. , 929 Holly Creek Drive, Great Falls, VA 27403 , US
O'BRIEN Alison D; Address - O'BRIEN, Alison, D. , 5514 Charlotte Road, Bethesda, MD 20817 , US
WACHTEL Marian R; Address - WACHTEL, Marian, R. , 18705 Walkers Choice
Road &2, Gaithersburg, MD 20879 , US
Patent and Priority Information (Country, Number, Date):
Patent: WO 9740161 A1 19971030
Application: WO 97US5832 19970418 (PCT/WO US9705832)
Priority Application: US 9615657 19960419; US 9615936 19960422
Designated States: AU; CA; JP; AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE;
IT; LU; MC; NL; PT; SE
Publication Language: English
Filing Language: English
Fulltext Word Count: 20670

3/3,KWIC/11 (Item 7 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00517416
THERAPEUTIC AND DIAGNOSTIC AGENTS FOR THE TREATMENT OF MICROBIAL INFECTIONS
AGENTS THERAPEUTIQUES ET DE DIAGNOSTIC POUR TRAITER LES INFECTIONS
MICROBIENNES
Patent Applicant/Assignee:
THE RESEARCH AND DEVELOPMENT INSTITUTE INC
PASCUAL David
BOND Clifford
BURRITT James
BURGESS Don
GLEE Pati
JUTILA John
JUTILA Mark
BARGATZE Robert
MCFETERS Gordon
PYLE Barry
CUTLER Jim E
HAN Yongmoon
Inventor(s):
PASCUAL David
BOND Clifford
BURRITT James
BURGESS Don
GLEE Pati
JUTILA John
JUTILA Mark
BARGATZE Robert
MCFETERS Gordon
PYLE Barry
CUTLER Jim E
HAN Yongmoon
Patent and Priority Information (Country, Number, Date):
Patent: WO 9718790 A2-A3 19970529
Application: WO 96US18796 19961121 (PCT/WO US9618796)
Priority Application: US 957477 19951122
Designated States: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CZ;
DE; DK; EE; ES; FI; GB; GE; HU; IL; IS; KG; KP; KR; KZ; LC; LK; LR; LS;
LT; LU; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SK; TJ;
TM; TR; TT; UA; UG; US; UZ; VN; KE; LS; MW; SD; SZ; UG; AM; AZ; BY; KG;
KZ; MD; RU; TJ; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL;
PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; NE; SN; TD; TG
Publication Language: English
Fulltext Word Count: 24126
Fulltext Availability:
Detailed Description
Claims

3/3,KWIC/12 (Item 8 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00384901
NOVEL POLYMERASE COMPOSITIONS AND USES THEREOF
NOUVELLES COMPOSITIONS A POLYMERASES ET LEURS UTILISATION
Patent Applicant/Assignee:
STRATAGENE
Inventor(s):
SORGE Joseph A
MULLINAX Rebecca Lynn
Patent and Priority Information (Country, Number, Date):
Patent: WO 9516028 A1 19950615
Application: WO 94US14065 19941207 (PCT/WO US9414065)
Priority Application: US 93164290 19931208; US 94197791 19940216
Designated States: CA; JP; AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU;
MC; NL; PT; SE
Publication Language: English

? ds
Set Items Description
S1 336 ((INTIMIN OR TIR)(20N)(SEQUENCE?)) AND COLI
S2 147 REMOVE DUPLICATES S1 (unique items)
S3 12 S2 AND ((94 OR 78 OR 94000 OR 78000)(10N)(KD OR KDA OR KIL-
ODALTON? OR DALTON?))

09/189415

********** STN Columbus **********

FILE 'HOME' ENTERED AT 18:51:31 ON 17 JUL 2000

=> file .bio
=> e finlay b/au
=> s e3-e16
L1 1288 ("FINLAY B"/AU OR "FINLAY B A"/AU OR "FINLAY B B"/AU OR "FINLAY B BRETT"/AU OR "FINLAY B BRETTT"/AU OR "FINLAY B F"/AU OR "FINLA Y B J"/AU OR "FINLAY B L"/AU OR "FINLAY B R"/AU OR "FINLAY BARBA RA L"/AU OR "FINLAY BLAND"/AU OR "FINLAY BLAND J"/AU OR "FINLAY BRETT"/AU OR "FINLAY BRETT B"/AU)

=> e kenny b/au
=> s e3,e13,e14,e6
L2 262 ("KENNY B"/AU OR "KENNY BRENDAN"/AU OR "KENNY BRENDAN G"/AU OR "KENNY B G"/AU)

=> e de vinney/au
=> s e4,e1
L3 5 ("DE VINNEY R"/AU OR "DE VINNA R S"/AU)

=> e devinney/au
=> s e1,e2,e14-e17
L4 63 ("DEVINNE R R R"/AU OR "DEVINNE R S C P I"/AU OR "DEVINNEY R"/AU OR "DEVINNEY R B"/AU OR "DEVINNEY R E"/AU OR "DEVINNEY REBEKAH" /AU)

=> e devinny r/au
=> e stein m/au
=> s e3,e41
L5 2020 ("STEIN M"/AU OR "STEIN MARCUS"/AU)

=> s (l1 or l2 or l3 or l4 or l5) and (intimin or hp90 or hp 90)
L6 69 (L1 OR L2 OR L3 OR L4 OR L5) AND (INTIMIN OR HP90 OR HP 90)

L7 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 1
AN 2000:438489 CAPLUS
TI Mechanical fractionation reveals structural requirements for enteropathogenic Escherichia coli Tir insertion into host membranes
AU Gauthier, Annick; De Grado, Myriam; ***Finlay, B. Brett***
CS Department of Biochemistry and Molecular Biology and Biotechnology Laboratory, University of British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Infect. Immun. (2000), 68(7), 4344-4348
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 31
RE
(1) Abe, A; Mol Microbiol 1999, V33, P1162 CAPLUS
(2) Bauer, M; Trends Cell Biol 2000, V10, P25 CAPLUS
(3) Collazo, C; Mol Microbiol 1997, V24, P747 CAPLUS
(4) DeVinney, R; Cell Mol Life Sci 1999, V55, P961 CAPLUS
(5) DeVinney, R; Infect Immun 1999, V67, P2389 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 21 SCISEARCH COPYRIGHT 2000 ISI (R)
AN 2000:475681 SCISEARCH
GA The Genuine Article (R) Number: 328VN
TI Crystal structure of enteropathogenic Escherichia coli ***intimin*** -receptor complex
AU Yu L; Frey E A; Pfuetzner R A; Creagh A L; Knoechel D G; Haynes C A; ***Finlay B B*** ; Strynadka N C J (Reprint)
CS UNIV BRITISH COLUMBIA, DEPT BIOCHEM & MOL BIOL, VANCOUVER, BC V6T 1Z3, CANADA (Reprint); UNIV BRITISH COLUMBIA, DEPT BIOCHEM & MOL BIOL, VANCOUVER, BC V6T 1Z3, CANADA; UNIV BRITISH COLUMBIA, BIOTECHNOL LAB, VANCOUVER, BC V6T 1Z3, CANADA
CYA CANADA
SO NATURE, (29 JUN 2000) Vol. 405, No. 6790, pp. 1073-1077. Publisher: MACMILLAN PUBLISHERS LTD, PORTERS SOUTH, 4 CRINAN ST, LONDON N1 9XW, ENGLAND. ISSN: 0028-0836.
DT Article; Journal
FS PHYS; LIFE; AGRI
LA English
REC Reference Count: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L7 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2000 ACS
AN 2000:421954 CAPLUS
TI Enteropathogenic E. coli translocated ***intimin*** receptor, Tir, interacts directly with .alpha.-actinin
AU Goosney, Danika L.; ***DeVinney, Rebekah*** ; Pfuetzner, Richard A.; Frey, Elizabeth A.; Strynadka, Natalie C.; ***Finlay, B. Brett***
CS Biotechnology Laboratory, The Department of Microbiology and Immunology, University of British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Curr. Biol. (2000), 10(12), 735-738
CODEN: CUBLE2; ISSN: 0960-9822
PB Elsevier Science Ltd.
DT Journal
LA English
RE.CNT 14
RE
(1) Abe, A; Mol Microbiol 1999, V33, P1162 CAPLUS
(2) deGrado, M; Cellular Microbiol 1999, V1, P7 CAPLUS
(3) Dramsi, S; Annu Rev Cell Dev Biol 1998, V14, P137 CAPLUS
(4) Frankel, G; Infect Immun 1995, V63, P4323 CAPLUS
(5) Frankel, G; J Biol Chem 1996, V271, P20359 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 2
AN 2000:240248 CAPLUS
DN 133:15368
TI Enteropathogenic Escherichia coli (EPEC) attachment to epithelial cells: exploiting the host cell cytoskeleton from the outside
AU Celli, Jean; Deng, Wanyin; ***Finlay, B. Brett***
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Cell. Microbiol. (2000), 2(1), 1-9
CODEN: CEMIF5; ISSN: 1462-5814
PB Blackwell Science Ltd.
DT Journal; General Review
LA English
RE.CNT 54
RE
(1) Abe, A; J Exp Med 1998, V188, P1907 CAPLUS
(2) Abe, A; Mol Microbiol 1999, V33, P1162 CAPLUS
(3) Adam, T; J Cell Biol 1995, V129, P367 CAPLUS
(4) Bain, C; Infect Immun 1998, V66, P3900 CAPLUS
(5) Baldwin, T; Infect Immun 1991, V59, P1599 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 3
AN 1999:326051 CAPLUS
DN 130:333761
TI Pathogenic Escherichia coli ***intimin*** receptor Tir and gene tir and methods for detecting gene tir or Tir protein and for drug screening
IN ***Finlay, B. Brett*** ; ***Kenny, Brendan*** ; ***Devinney,*** *** Rebekah*** ; ***Stein, Marcus***
PA University of British Columbia, Can.
SO PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9924576	A1	19990520	WO 1998-CA1042	19981110

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU,

CZ, DE,
DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 9911373 A1 19990531 AU 1999-11373 19981110
PRAI US 1997-65130 19971112
WO 1998-CA1042 19981110
RE.CNT 6
RE
(1) Deibel, C; Molecular Microbiology 1998, V28(3), P463 CAPLUS
(2) Kenny, B; Cell 1997, V91, P511 CAPLUS
(3) Kenny, B; Infection and Immunity 1997, V65(7), P2528 CAPLUS
(4) Paton, A; Database EMBL - EMPRO 1998
(5) Paton, A; Infection and Immunity 1998, V66(11), P5580 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 6 OF 21 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1999-540860 [45] WPIDS
DNN N1999-400815 DNC C1999-158073
TI Identifying antibacterial agents that inhibit the Gram-negative type III secretion system, for treating infections - by screening for inhibition of virulence factors secreted by this system.
DC B04 D16 S03
IN ***FINLAY, B B*** ; ***KENNY, B*** ; ***STEIN, M***
PA (UYBR-N) UNIV BRITISH COLUMBIA
CYC 83
PI WO 9945136 A1 19990910 (199945)* EN 51p C12Q001-02
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD
GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT
UA UG UZ VN YU ZW
AU 9932431 A 19990920 (200007) C12Q001-02
ADT WO 9945136 A1 WO 1999-CA183 19990305; AU 9932431 A AU 1999-32431 19990305
FDT AU 9932431 A Based on WO 9945136
PRAI US 1998-76980 19980305
IC ICM C12Q001-02
ICS C12Q001-32; C12Q001-34; C12Q001-42; C12Q001-48; C12Q001-66; G01N033-68

L7 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 4
AN 1999:291203 CAPLUS
DN 131:85390
TI Enterohemorrhagic Escherichia coli O157:H7 produces Tir, which is translocated to the host cell membrane but is not tyrosine phosphorylated
AU ***DeVinney, Rebekah*** ; Stein, Markus; Reinscheid, Dieter; Abe, Akio;
Ruschkowski, Sharon; ***Finlay, B. Brett***
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
V6T 1ZA, Can.
SO Infect. Immun. (1999), 67(5), 2389-2398
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 45
RE
(1) Abe, A; Infect Immun 1997, V65, P3547 CAPLUS
(4) Bieber, D; Science 1998, V280, P2114 CAPLUS
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(7) Deibel, C; Mol Microbiol 1998, V28, P463 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 5
AN 1999:159076 CAPLUS
DN 130:308856
TI Phosphorylation of tyrosine 474 of the enteropathogenic Escherichia coli (EPEC) Tir receptor molecule is essential for actin nucleating activity and is preceded by additional host modifications
AU ***Kenny, Brendan***
CS Department of Pathology and Microbiology, School of Medical Sciences, University Walk, Bristol, BS8 1TD, UK
SO Mol. Microbiol. (1999), 31(4), 1229-1241
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 33
RE
(1) Abe, A; Infect Immun 1997, V65, P3547 CAPLUS
(2) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
(3) Anderson, D; Science 1997, V278, P1140 CAPLUS
(4) Beaulieu, J; J Cell Sci 1992, V102, P427 CAPLUS
(5) Clark, M; Infect Immun 1998, V66, P1237 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 6
AN 1999:632013 CAPLUS
DN 131:333662
TI Enteropathogenic Escherichia coli translocated ***intimin*** receptor, Tir, requires a specific chaperone for stable secretion
AU Abe, Akio; De Grado, Myriam; Pfuetzner, Richard A.; Sanchez-SanMartin,
Claudia; ***DeVinney, Rebekah*** ; Puente, Jose Luis; Strynadka, Natalie C. J.; ***Finlay, B. Brett***
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
V6T 1Z3, Can.
SO Mol. Microbiol. (1999), 33(6), 1162-1175
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 57
RE
(1) Abe, A; J Exp Med 1998, V188, P1907 CAPLUS
(2) An, H; FEMS Microbiol Lett 1997, V148, P239 CAPLUS
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(4) Beaudry, M; J Clin Microbiol 1996, V34, P144 CAPLUS
(6) Cheng, L; Mol Microbiol 1997, V24, P757 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 7
AN 1999:422677 CAPLUS
TI Enteropathogenic Escherichia coli. A pathogen that inserts its own receptor into host cells
AU ***De Vinney, R.*** ; Gauthier, A.; Abe, A.; ***Finlay, B. B.***
CS Biotechnology Laboratory, Univ. British Columbia, Vancouver, BC, V6T 1Z4,
Can.
SO Cell. Mol. Life Sci. (1999), 55(6/7), 961-976
CODEN: CMLSFI; ISSN: 1420-682X
PB Birkhaeuser Verlag
DT Journal
LA English
RE.CNT 122
RE
(1) Abe, A; Infect Immun 1997, V65, P3547 CAPLUS
(3) Adams, L; Infect Immun 1997, V65, P5222 CAPLUS
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(5) Allaoui, A; Mol Microbiol 1995, V18, P343 CAPLUS
(6) Anderson, D; Science 1997, V278, P1140 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 8
AN 1999:83261 CAPLUS
DN 130:236380
TI Enteropathogenic Escherichia coli inhibits phagocytosis

AU Goosney, Danika L.; Celli, Jean; ***Kenny, Brendan*** ; ***Finlay, B.***
*** Brett***
CS Biotechnology Laboratory and Departments of Microbiology & Immunology and
of Biochemistry & Molecular Biology, University of British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Infect. Immun. (1999), 67(2), 490-495
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 31
RE
(1) Abe, A; Infect Immun 1997, V65, P3547 CAPLUS
(2) Andersson, K; Mol Microbiol 1996, V20, P1057 CAPLUS
(3) Baldwin, T; Infect Immun 1991, V59, P1599 CAPLUS
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(6) Donnenberg, M; Infect Immun 1990, V58, P1565 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2000 ACS
AN 2000:298095 CAPLUS
TI Identification of the ***intimin*** -binding domain of Tir of enteropathogenic Escherichia coli
AU De Grado, Myriam; Abe, Akio; Gauthier, Annick; Steele-Mortimer, Olivia;
DeVinney, Rebekah ; ***Finlay, B. Brett***
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
V6T 1Z3, Can.
SO Cell. Microbiol. (1999), 1(1), 7-17
CODEN: CEMIF5; ISSN: 1462-5814
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 27
RE
(1) DeVinney, R; Infect Immun 1999, V67, P2389 CAPLUS
(3) Donnenberg, M; Infect Immun 1991, V59, P4310 CAPLUS
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(5) Donnenberg, M; J Clin Invest 1993, V92, P1412 CAPLUS
(6) Elliott, S; Mol Microbiol 1998, V28, P1 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 9
AN 1997:408570 CAPLUS
DN 127:134079
TI ***Intimin*** -dependent binding of enteropathogenic Escherichia coli to host cells triggers novel signaling events, including tyrosine phosphorylation of phospholipase C-.gamma.1
AU ***Kenny, Brendan*** ; ***Finlay, B. Brett***
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
V6T 1Z3, Can.
SO Infect. Immun. (1997), 65(7), 2528-2536
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English

L7 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 10
AN 1997:755652 CAPLUS
DN 128:72707
TI Enteropathogenic E. coli (EPEC) transfers its receptor for intimate adherence into mammalian cells
AU ***Kenny, Brendan*** ; ***DeVinney, Rebekah*** ; Stein, Markus;
Reinscheid, Dieter J.; Frey, Elizabeth A.; ***Finlay, B. Brett***
CS Biotechnol. Lab., Dep. Biochem. Mol. Biol., Dep. Microbiol. Immunology,
Univ. British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Cell (Cambridge, Mass.) (1997), 91(4), 511-520
CODEN: CELLB5; ISSN: 0092-8674
PB Cell Press
DT Journal
LA English

L7 ANSWER 15 OF 21 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:187776 BIOSIS
DN PREV199799486979
TI Enteropathogenic E. coli exploitation of host epithelial cells.
AU ***Finlay, B. Brett (1)*** ; Ruschkowski, Sharon; ***Kenny,*** *** Brendan*** ; Stein, Markus; Reinscheid, Dieter J.; Stein, Murry A.; Rosenshine, Ilan
CS (1) Biotechnol. Lab., Univ. B.C., Vancouver, BC V6T 1Z3 Canada
SO Ades, E. W. [Editor]; Morse, S. A. [Editor]; Rest, R. F. [Editor]. Annals of the New York Academy of Sciences, (1996) Vol. 797, pp. 26-31. Annals of
the New York Academy of Sciences; Microbial pathogenesis and immune response, II.
Publisher: New York Academy of Sciences 2 East 63rd Street, New York, New
York 10021, USA.
Meeting Info.: Conference New York, New York, USA October 25-28, 1995
ISSN: 0077-8923. ISBN: 1-57331-017-4 (paper), 1-57331-016-6 (cloth).
DT Book; Conference
LA English

L7 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 11
AN 1996:381158 CAPLUS
DN 125:53467
TI A pathogenic bacterium triggers epithelial signals to form a functional bacterial receptor that mediates actin pseudopod formation
AU Rosenshine, Ilan; Ruschkowski, Sharon; Stein, Markus; Reinscheid, Dieter
J.; Mills, Scott D.; ***Finlay, B. Brett***
CS Department Biotechnology and Molecular Genetics, Hebrew University, Jerusalem, 12272, Israel
SO EMBO J. (1996), 15(11), 2613-2624
CODEN: EMJODG; ISSN: 0261-4189
DT Journal
LA English

L7 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 12
AN 1996:129653 CAPLUS
DN 124:226331
TI Expression of attaching/effacing activity by enteropathogenic Escherichia coli depends on growth phase, temperature, and protein synthesis upon contact with epithelial cells
AU Rosenshine, Ilan; Ruschkowski, Sharon; ***Finlay, B. Brett***
CS Fac. Medicine, Hebrew Univ., Jerusalem, 91120, Israel
SO Infect. Immun. (1996), 64(3), 966-73
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English

L7 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 13
AN 1996:286497 CAPLUS
DN 125:2537
TI EspA, a protein secreted by enteropathogenic Escherichia coli, is required to induce signals in epithelial cells
AU ***Kenny, Brendan*** ; Lai, Li-Ching; ***Finlay, B. Brett*** ; Donnenberg, Michael S.
CS Dep of Biochemistry and Molecular Biology, Univ. of British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO Mol. Microbiol. (1996), 20(2), 313-323
CODEN: MOMIEE; ISSN: 0950-382X
DT Journal
LA English

L7 ANSWER 19 OF 21 MEDLINE DUPLICATE 14
AN 97146492 MEDLINE
DN 97146492
TI Enteropathogenic E. coli exploitation of host epithelial cells.
AU ***Finlay B B*** ; Ruschkowski S; ***Kenny B*** ; ***Stein M*** ;
Reinscheid D J; Stein M A; Rosenshine I
CS Biotechnology Laboratory, University of British Columbia, Vancouver, Canada.. bfinlay@unixg.ubc.ca
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1996 Oct 25) 797 26-31. Ref:

51
Journal code: 5NM. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals; Cancer Journals
EM 199704
EW 19970402

L7 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2000 ACS
AN 1994:160431 CAPLUS
DN 120:160431
TI A diarrheal pathogen, enteropathogenic Escherichia coli (EPEC), triggers a flux of inositol phosphates in infected epithelial cells
AU Foubister, Vida; Rosenshine, Ilan; ***Finlay, B. Brett***
CS Biotechnol. Lab., Univ. British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO J. Exp. Med. (1994), 179(3), 993-8
CODEN: JEMEAV; ISSN: 0022-1007
DT Journal
LA English

L7 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 15
AN 1992:609978 CAPLUS
DN 117:209978
TI Signal transduction between enteropathogenic Escherichia coli (EPEC) and epithelial cells: EPEC induces tyrosine phosphorylation of host cell proteins to initiate cytoskeletal rearrangement and bacterial uptake
AU Rosenshine, Ilan; Donnenberg, Michael S.; Kaper, James B.; ***Finlay, B.***
*** Brett***
CS Dep. Biochem., Univ. British Columbia, Vancouver, BC, V6T 1Z3, Can.
SO EMBO J. (1992), 11(10), 3551-60
CODEN: EMJODG; ISSN: 0261-4189
DT Journal
LA English

=> d l7 1-21 ti,ab
L7 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 1
TI Mechanical fractionation reveals structural requirements for enteropathogenic Escherichia coli Tir insertion into host membranes
AB Enteropathogenic Escherichia coli (EPEC) inserts its receptor for intimate adherence (Tir) into host cell membranes by using a type III secretion system. Detergents are frequently used to fractionate infected host cells to investigate bacterial protein delivery into mammalian cells. In this study, we found that the Triton X-100-sol. membrane fraction from EPEC-infected HeLa cells was contaminated with bacterial proteins. We therefore applied a mech. method of cell lysis and ultracentrifugation to fractionate infected HeLa cells to investigate the biol. and biochem. of Tir delivery and translocation. This method demonstrates that the translocation of Tir into the host cell membrane requires its transmembrane domains, but not tyrosine phosphorylation or binding to Tir's ligand, ***intimin*** .

L7 ANSWER 2 OF 21 SCISEARCH COPYRIGHT 2000 ISI (R)
TI Crystal structure of enteropathogenic Escherichia coli ***intimin*** -receptor complex
AB ***Intimin*** and its translocated ***intimin*** receptor (Tir) are bacterial proteins that mediate adhesion between mammalian cells and attaching and effacing (A/E) pathogens. Enteropathogenic Escherichia coli (EPEC) causes significant paediatric morbidity and mortality world-wide(1). A related A/E pathogen, enterohaemorrhagic E. coli (EHEC; O157:H7) is one of the most important food-borne pathogens in North America, Europe and Japan. A unique and essential feature of A/E bacterial pathogens is the formation of actin-rich pedestals beneath the intimately adherent bacteria and localized destruction of the intestinal brush border(2). The bacterial outer membrane adhesin, ***intimin*** (3), is necessary for the production of the A/E lesion and diarrhoea(4). The A/E bacteria translocate their own receptor for ***intimin*** , Tir(5), into the membrane of mammalian cells using the type III secretion system. The translocated Tir triggers additional host signalling events and actin nucleation, which are essential for lesion formation. Here we describe the the crystal structures of an EPEC ***intimin*** carboxyterminal fragment alone and in complex with the EPEC Tir ***intimin*** -binding domain, giving insight into the molecular mechanisms of adhesion of A/E pathogens.

L7 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2000 ACS
TI Enteropathogenic E. coli translocated ***intimin*** receptor, Tir, interacts directly with .alpha.-actinin
AB Enteropathogenic Escherichia coli (EPEC) triggers a dramatic rearrangement
of the host epithelial cell actin cytoskeleton to form an attaching and effacing lesion, or pedestal. The pathogen remains attached extracellularly to the host cell through the pedestal for the duration of the infection. At the tip of the pedestal is a bacterial protein, Tir, which is secreted from the bacterium into the host cell plasma membrane, where it functions as the receptor for an EPEC outer membrane protein, ***intimin*** [1]. Delivery of Tir to the host cell results in its tyrosine phosphorylation, followed by Tir- ***intimin*** binding. Tir is believed to anchor EPEC firmly to the host cell, although its direct linkage to the cytoskeleton is unknown. Here, we show that Tir directly binds the cytoskeletal protein .alpha.-actinin. .alpha.-Actinin is recruited to the pedestal in a Tir-dependent manner and colocalizes with Tir in infected host cells. Binding is mediated through the amino terminus of Tir. Recruitment of .alpha.-actinin occurs independently of Tir tyrosine phosphorylation. Recruitment of actin, VASP, and N-WASP, however, is abolished in the absence of this tyrosine phosphorylation. These results suggest that Tir plays at least three roles in the host cell during infection: binding ***intimin*** on EPEC; mediating a stable anchor with .alpha.-actinin through its amino terminus in a phosphotyrosine-independent manner; and recruiting addnl. cytoskeletal proteins at the carboxyl terminus in a phosphotyrosine-dependent manner. These findings demonstrate the first known direct linkage between extracellular EPEC, through the transmembrane protein Tir, to the host cell actin cytoskeleton via .alpha.-actinin.

L7 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 2
TI Enteropathogenic Escherichia coli (EPEC) attachment to epithelial cells: exploiting the host cell cytoskeleton from the outside
AB A review, with 54 refs. Enteropathogenic Escherichia coli (EPEC), a leading cause of human infantile diarrhea, is the prototype for a family of intestinal bacterial pathogens that induce attaching and effacing (A/E) lesions on host cells. A/E lesions are characterized by localized effacement of the brush border of enterocytes, intimate bacterial attachment and pedestal formation beneath the adherent bacteria. As a result of some recent breakthrough discoveries, EPEC has now emerged as a fascinating paradigm for the study of host-pathogen interactions and cytoskeletal rearrangements that occur at the host cell membrane. EPEC uses a type III secretion machinery to attach to epithelial cells, translocating its own receptor for intimate attachment, Tir, into the host cell, which then binds to ***intimin*** on the bacterial surface. Studies of EPEC-induced cytoskeletal rearrangements have begun to provide
clues as to the mechanisms used by this pathogen to subvert the host cell cytoskeleton and signaling pathways. These findings have unraveled new ways by which pathogenic bacteria exploit host processes from the cell surface and have shed new light on how EPEC might cause diarrhea.

L7 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 3
TI Pathogenic Escherichia coli ***intimin*** receptor Tir and gene tir and methods for detecting gene tir or Tir protein and for drug screening
AB A polypeptide, called Tir (for translocated ***intimin*** receptor), which is secreted by attaching and effacing pathogens, such as the enteropathogenic (EPEC) and enterohemorrhagic (EHEC) E. coli is disclosed.
These bacterial pathogens insert their own receptors into mammalian cell surfaces, to which the bacterial pathogen then adheres to trigger addnl. host signaling events and actin nucleation. Diagnosis of disease caused by pathogenic E. coli can be performed by the use of antibodies which bind to Tir to detect the protein or the use of nucleic acid probes for detection of nucleic acids encoding Tir polypeptide. Isolated nucleic acid sequences encoding Tir polypeptide, Tir peptides, a recombinant method for producing recombinant Tir, antibodies which bind to Tir, and a kit for the detection of Tir-producing E. coli are provided. A method of immunizing a host with Tir to induce a protective immune response to Tir or a second polypeptide of interest is also provided. A method for screening for compds. which interfere with the binding of bacterial pathogens to their receptors is further provided. Thus, protein ***Hp90*** , previously believed to be a host membrane protein, has been

identified as an EHEC- or EPEC-secreted protein which acts as an ***intimin*** receptor. Proteins encoded by the espA and espB genes were necessary for delivery of Tir to the host membrane.

L7 ANSWER 6 OF 21 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD

TI Identifying antibacterial agents that inhibit the Gram-negative type III secretion system, for treating infections - by screening for inhibition of virulence factors secreted by this system.

AB WO 9945136 A UPAB: 19991103

NOVELTY - Identification of antibacterial agents (I) comprises:

(i) treating bacteria that contain a polynucleotide (II) which encodes a polypeptide (III) secreted by the type III secretion system (3SS) with a test compound and

(ii) detecting secretion of (III).

A reduction of secretion, relative to that in bacteria not treated with the test compound, indicates an inhibitor of 3SS.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a kit containing, in separate containers, the bacteria and a system for detecting secretion of (III).

ACTIVITY - Antibacterial.

MECHANISM OF ACTION - (I) blocks the 3SS which is used to secrete virulence factors essential for pathogenicity.

USE - (I) are used:

(i) to treat bacterial infections in humans, other animals and plants, e.g. where caused by enteropathogenic or enterohemorrhagic Escherichia coli, Yersinia species, Shigella species, Pseudomonas aeruginosa, P. syringae, Xanthomonas campestris, or many others listed;

(ii) for analyzing the functional mechanisms of 3SS (which involves about 20 gene products), and

(iii) for development of more powerful or specific inhibitors.

ADVANTAGE - 3SS is conserved in many Gram-negative bacteria, so (I) should have a broad spectrum of activity, and is used exclusively to secrete virulence factors. It is absent from non-pathogenic bacteria. The method can identify inhibitors specific for 3SS.

Dwg.0/2

L7 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 4

TI Enterohemorrhagic Escherichia coli O157:H7 produces Tir, which is translocated to the host cell membrane but is not tyrosine phosphorylated

AB Intimate attachment to the host cell leading to the formation of attaching and effacing (A/E) lesions is an essential feature of enterohemorrhagic Escherichia coli (EHEC) O157:H7 pathogenesis. In a related pathogen, enteropathogenic E. coli (EPEC), this activity is dependent upon translocation of the ***intimin*** receptor, Tir, which becomes tyrosine phosphorylated within the host cell membrane. In contrast, the accumulation of tyrosine-phosphorylated proteins beneath adherent EHEC bacteria does not occur, leading to questions about whether EHEC uses a Tir-based mechanism for adherence and A/E lesion formation. In this report, we demonstrate that EHEC produces a functional Tir that is inserted into host cell membranes, where it serves as an ***intimin*** receptor. However, unlike in EPEC, in EHEC Tir is not tyrosine phosphorylated yet plays a key role in both bacterial adherence to epithelial cells and pedestal formation. EHEC, but not EPEC, was unable to synthesize Tir in Luria-Bertani medium but was able to secrete Tir into M9 medium, suggesting that Tir synthesis and secretion may be regulated differently in these two pathogens. EHEC Tir and EPEC Tir both bind ***intimin*** and focus cytoskeletal rearrangements, indicating that tyrosine phosphorylation is not needed for pedestal formation. EHEC and EPEC intimins are functionally interchangeable, but EHEC Tir shows a much greater affinity for EHEC ***intimin*** than for EPEC ***intimin*** . These findings highlight some of the differences and similarities between EHEC and EPEC virulence mechanisms, which can be exploited to further define the mol. basis of pedestal formation.

L7 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 5

TI Phosphorylation of tyrosine 474 of the enteropathogenic Escherichia coli (EPEC) Tir receptor molecule is essential for actin nucleating activity and is preceded by additional host modifications

AB The enteropathogenic Escherichia coli (EPEC) Tir protein becomes tyrosine phosphorylated in host cells and displays an increase in apparent mol. mass. The interaction of Tir with the EPEC outer membrane protein, ***intimin*** , triggers actin nucleation beneath the adherent bacteria. The enterohaemorrhagic E. coli 0157:H7 (EHEC) Tir mol. is not tyrosine phosphorylated. In this paper, Tir tyrosine phosphorylation is shown to be essential for actin nucleation activity, but not for the increase in apparent mol. mass obsd. in target cells. Tyrosine phosphorylation had no role in Tir mol. mass shift, indicating addnl. host modifications. Anal. of Tir intermediates indicates that tyrosine-independent modification functions to direct Tir's correct insertion from the cytoplasm into the host membrane. Deletion anal. identified Tir domains participating in translocation, assocn. with the host membrane, modification and antibody recognition. ***Intimin*** was found to bind a 55-amino-acid region (TIBA) within Tir that topol. and sequence anal. suggests is located in an extracellular loop. Homologous TIBA sequences exist in integrins, which also bind ***intimin*** . Collectively, this study provides definitive evidence for the importance of tyrosine phosphorylation for EPEC Tir function and reveals differences in the pathogenicity of EPEC and EHEC. The data also suggest a mechanism for Tir insertion into the host membrane, as well as providing clues to the mode of ***intimin*** -integrin interaction.

L7 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 6

TI Enteropathogenic Escherichia coli translocated ***intimin*** receptor, Tir, requires a specific chaperone for stable secretion

AB Enteropathogenic Escherichia coli (EPEC) secretes several Esps (E. coli-secreted proteins) that are required for full virulence. Insertion of the bacterial protein Tir into the host epithelial cell membrane is facilitated by a type III secretion app., and at least EspA and EspB are required for Tir translocation. An EPEC outer membrane protein, ***intimin*** , interacts with Tir on the host membrane to establish intimate attachment and formation of a pedestal-like structure. In this study, we identified a Tir chaperone, CesT, whose gene is located between tir and eae (which encodes ***intimin***). A mutation in cesT abolished Tir secretion into culture supernatants and significantly decreased the amt. of Tir in the bacterial cytoplasm. In contrast, this mutation did not affect the secretion of the Esp proteins. The level of tir mRNA was not affected by the cesT mutation, indicating that CesT acts at the post-transcriptional level. The cesT mutant could not induce host cytoskeletal rearrangements, and displayed the same phenotype as the tir mutant. Gel overlay and GST pulldown assays demonstrated that CesT specifically interacts with Tir, but not with other Esp proteins. Furthermore, by using a series of Tir deletion derivs., we detd. that the CesT binding domain is located within the first 100 amino-terminal residues of Tir, and that the pool of Tir in the bacterial cytoplasm was greatly reduced when this domain was disrupted. Interestingly, this domain was not sufficient for Tir secretion, and at least the first 200 residues of Tir were required for efficient secretion. Gel filtration studies showed that Tir-CesT forms a large multimeric complex. Collectively, these results indicate that CesT is a Tir chaperone that may act as an anti-degrdn. factor by specifically binding to its amino-terminus, forming a multimeric stabilized complex.

L7 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 7

TI Enteropathogenic Escherichia coli. A pathogen that inserts its own receptor into host cells

AB Enteropathogenic Escherichia coli (EPEC) is a major cause of infant diarrhea, killing hundreds of thousands of children per yr worldwide. Intimate attachment to the host cell leading to the formation of actin-rich pedestals beneath the adhering bacteria is an essential feature of EPEC pathogenesis. EPEC attaches to host cells via the outer membrane adhesin, ***intimin*** . It was recently shown that EPEC inserts its own receptor for intimate adherence, Tir (translocated ***intimin*** receptor) into the host cell membrane. The focus of this review is on the discovery and characterization of this novel receptor, and our current understanding of its role in pedestal formation. Gram-neg. bacterial secretion systems, including type III secretion systems, are reviewed and discussed in the context of Tir delivery into the host cell membrane. The relationship and relevance of in vitro models compared to the actual in viva situation is essential to understanding disease. We have critically reviewed the use of animal models in studying EPEC infection. Elucidating the function of Tir will contribute to our understanding of how EPEC mediates disease.

L7 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 8

TI Enteropathogenic Escherichia coli inhibits phagocytosis

AB Enteropathogenic Escherichia coli (EPEC) interacts with intestinal epithelial cells, activating host signaling pathways leading to

cytoskeletal rearrangements and ultimately diarrhea. Here it is shown that EPEC interacts with the macrophage-like cell line J774A.1 to inhibit phagocytosis by these cells. Antiphagocytic activity was also obsd. in cultured RAW macrophage-like cells upon EPEC infection. The EPEC antiphagocytic phenotype was dependent on the type III secretion pathway of EPEC and its secreted proteins, including EspA, EspB, and EspD. ***Intimin*** and Tir mutants displayed intermediate antiphagocytic activity, suggesting that intimate attachment mediated by ***intimin*** -Tir binding may also play a role in antiphagocytosis. Tyrosine dephosphorylation of several host proteins was obsd. following infection with secretion-competent EPEC but not with secretion-deficient mutants. Dephosphorylation was detectable 120 min after infection with EPEC, directly correlating with the onset of the antiphagocytic phenotype. Inhibition of protein tyrosine phosphatases by pervanadate treatment increased the no. of intracellular wild-type EPEC organisms to levels seen with secretion-deficient mutants, suggesting that dephosphorylation events are linked to the antiphagocytic phenotype. No tyrosine phosphatase activity was detected with the EPEC-secreted proteins, suggesting that EPEC induces antiphagocytosis via a different mechanism than Yersinia species. The present findings demonstrate a novel function for EPEC-secreted proteins in triggering macrophage protein tyrosine dephosphorylation and inhibition of phagocytosis.

L7 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2000 ACS
TI Identification of the ***intimin*** -binding domain of Tir of enteropathogenic Escherichia coli
AB Enteropathogenic Escherichia coli (EPEC) attaches intimately to mammalian
cells via a bacterial outer membrane adhesion mol., ***intimin*** , and its receptor in the host cell membrane, Tir. Tir is a bacterial protein translocated into the host cell membrane and tyrosine phosphorylated after insertion. Tir- ***intimin*** binding induces organized actin polymn. beneath the adherent bacteria, resulting in the formation of pedestal-like structures. A series of Tir deletion derivs. were constructed to analyze which Tir domains are involved in ***intimin*** binding. We have localized the ***intimin*** -binding domain (IBD) of Tir using a yeast two-hybrid system and a gel-overlay approach to a region of 109 amino acids that is predicted to be exposed on the surface of the plasma membrane. A truncated Tir protein lacking this domain was translocated to the host cell membrane and tyrosine phosphorylated, but failed to bind ***intimin*** or to induce either actin polymn. or Tir accumulation beneath the bacteria. These results indicate that only a small region of Tir is needed to bind ***intimin*** and support the predicted topol. for Tir, with both N- and C-terminal regions in the mammalian cell cytosol. They also confirm that Tir- ***intimin*** interactions are needed for cytoskeletal organization. We have also identified N-terminal regions involved in Tir stability and Tir secretion to the media.

L7 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 9
TI ***Intimin*** -dependent binding of enteropathogenic Escherichia coli to host cells triggers novel signaling events, including tyrosine phosphorylation of phospholipase C-.gamma.1
AB Enteropathogenic Escherichia coli (EPEC) interactions with HeLa epithelial
cells induced the tyrosine phosphorylation of a host protein of approx. 150 kDa, Hp150. Phosphorylation of this protein band was dependent on the
interaction of the EPEC protein ***intimin*** with epithelial cell surfaces and was correlated with pedestal formation. Hp150 phosphorylation was specifically inhibited by the addn. of cytochalasin D, an inhibitor of actin polymn., although this appeared to be an indirect effect preventing interaction of ***intimin*** with its receptor, tyrosine-phosphorylated ***Hp90*** , and thus triggering Hp150 phosphorylation. This suggests the involvement of an actin-based movement
of membrane-bound tyrosine-phosphorylated ***Hp90*** to allow its interaction with ***intimin*** . Anal. of the tyrosine-phosphorylated Hp150 protein demonstrated that it is heterogeneous in compn., with phospholipase C-.gamma.1 (PLC-.gamma.1) being a minor component. Activation of PLC-.gamma.1 by tyrosine phosphorylation leads to inositol triphosphate and Ca2+ fluxes, events detected following EPEC infection. EPEC also induced tyrosine dephosphorylation of host proteins, including a 240-kDa host protein (Hp240), following EPEC infection. Protein dephosphorylation appears to be a signaling event which occurs independently of ***intimin*** . Inhibition of host tyrosine dephosphorylation events by the addn. of the tyrosine phosphatase inhibitor sodium vanadate did not prevent actin accumulation beneath the adherent bacteria. The authors conclude that EPEC induces two sets of signaling events following infection. One set is dependent on EPEC proteins secreted by the type III secretion pathway (EspA and EspB) which induces ***Hp90*** tyrosine phosphorylation and dephosphorylation of host phosphotyrosine proteins. The second set, which is also dependent on the first signaling events, requires ***intimin*** interaction with its receptor, tyrosine-phosphorylated ***Hp90*** , to trigger Hp150 and PLC-.gamma.1 tyrosine phosphorylation as well as pedestal formation. The second set, which is also dependent on the first signaling events, requires ***intimin*** interaction with its receptor, tyrosine-phosphorylated ***Hp90*** , to trigger Hp150 and PLC-.gamma.1
tyrosine phosphorylation as well as pedestal formation.

L7 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 10
TI Enteropathogenic E. coli (EPEC) transfers its receptor for intimate adherence into mammalian cells
AB Enteropathogenic Escherichia coli (EPEC) belongs to a group of bacterial pathogens that induce epithelial cell actin rearrangements resulting in pedestal formation beneath adherent bacteria. This requires the secretion of specific virulence proteins needed for signal transduction and intimate adherence. EPEC interaction induces tyrosine phosphorylation of a protein in the host membrane, ***Hp90*** , which is the receptor for the EPEC outer membrane protein, ***intimin*** . ***Hp90*** - ***intimin***
interaction is essential for intimate attachment and pedestal formation. Here, we demonstrate that ***Hp90*** is actually a bacterial protein (Tir). Thus, this bacterial pathogen inserts its own receptor into mammalian cell surfaces, to which it then adheres to trigger addnl. host signaling events and actin nucleation. It is also tyrosine-phosphorylated upon transfer into the host cell.

L7 ANSWER 15 OF 21 BIOSIS COPYRIGHT 2000 BIOSIS
TI Enteropathogenic E. coli exploitation of host epithelial cells.

L7 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 11
TI A pathogenic bacterium triggers epithelial signals to form a functional bacterial receptor that mediates actin pseudopod formation
AB Enteropathogenic Escherichia coli (EPEC) belongs to a group of bacterial pathogens that induce actin accumulation beneath adherent bacteria. We found that EPEC adherence to epithelial cells mediates the formation of finger-like pseudopods (up to 10 .mu.m) beneath bacteria. These actin-rich structures also contain tyrosine phosphorylated host proteins concd. at the pseudopod tip beneath adherent EPEC. Intimate bacterial adherence (and pseudopod formation) occurred only after prior bacterial induction of tyrosine phosphorylation of an epithelial membrane protein, ***Hp90*** , which then assocs. directly with an EPEC adhesin, ***intimin*** . These interactions lead to cytoskeletal nucleation and pseudopod formation. This is the first example of a bacterial pathogen that triggers signals in epithelial cells which activates receptor binding activity to a specific bacterial ligand and subsequent cytoskeletal rearrangement.

L7 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 12
TI Expression of attaching/effacing activity by enteropathogenic Escherichia coli depends on growth phase, temperature, and protein synthesis upon contact with epithelial cells
AB Enteropathogenic Escherichia coli (EPEC) induces tyrosine phosphorylation
of a 90-kDa protein (***Hp90***) in infected epithelial cells. This in turn facilitates intimate binding of EPEC via the outer membrane protein ***intimin*** , effacement of host cell microvilli, cytoskeletal rearrangement, and bacterial uptake. This phenotype has been commonly referred to as attaching/effacing (A/E). The ability of EPEC to induce A/E lesions was dependent on bacterial growth phase and temp. Early-logarithmic-phase EPEC grown at 37.degree. elicits strong A/E activity within minutes after infection of HeLa epithelial cells. EPEC de novo protein synthesis during the first minutes of interaction with the host cell was required to elicit A/E lesions. However, once formed, bacterial viability was not needed to maintain A/E lesions. The type of growth media and partial O2 pressure level do not seem to affect the ability of EPEC to cause A/E lesions. These results indicates that the A/E activity of EPEC is tightly regulated by environmental and host factors.

L7 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 13

TI EspA, a protein secreted by enteropathogenic Escherichia coli, is required to induce signals in epithelial cells

AB Enteropathogenic Escherichia coli (EPEC) is a leading cause of infant diarrhea. EPEC mediates several effects on host epithelial cells, including activation of signal-transduction pathways, cytoskeletal rearrangement along with pedestal and attaching/effacing lesion formation. It has been previously shown that the EPEC eaeB (espB) gene encodes a secreted protein required for signal transduction and adherence, while eaeA encodes ***intimin*** , an EPEC membrane protein that mediates intimate adherence and contributes to focusing of cytoskeletal proteins beneath bacteria. DNA-sequence anal. of a region between eaeA and eaeB identified a predicted open reading frame (espA) that matched the amino-terminal sequence of a 25 kDa EPEC secreted protein. A mutant with a non-polar insertion in espA does not secrete this protein, activate epithelial cell signal transduction or cause cytoskeletal rearrangement. These phenotypes were complemented by a cloned espA gene. The espA mutant is also defective for invasion. It is concluded that espA encodes an EPEC secreted protein that is necessary for activating epithelial signal transduction, intimate contact, and formation of attaching and effacing lesions, processes which are central to pathogenesis.

L7 ANSWER 19 OF 21 MEDLINE DUPLICATE 14

TI Enteropathogenic E. coli exploitation of host epithelial cells.

AB Enteropathogenic E. coli (EPEC) is a leading cause of neonatal diarrhea worldwide. These organisms adhere to the intestinal cell surface, causing rearrangement in the epithelial cell surface and underlying cytoskeleton, resulting in a structure termed an attaching/effacing (A/E) lesion. A/E lesion formation is thought necessary for EPEC-mediated disease. EPEC secretes several proteins that trigger signal transduction, intimate adherence, and cytoskeletal rearrangements in epithelial cells. Additionally, it produces ***intimin*** , an outer membrane product that mediates intimate adherence. Together these various bacterial molecules contribute to the intimate relationship that is formed by EPEC with host epithelial cells which results in A/E lesion formation and diarrhea.

L7 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2000 ACS

TI A diarrheal pathogen, enteropathogenic Escherichia coli (EPEC), triggers a flux of inositol phosphates in infected epithelial cells

AB Enteropathogenic Escherichia coli (EPEC) is a bacterial pathogen that causes diarrhea in infants by adhering to intestinal epithelial cells. EPEC induces host cell protein phosphorylation and increases intracellular calcium levels that may function to initiate cytoskeletal rearrangement. The authors found that EPEC triggers the release of inositol phosphates (IPs) after adherence of bacteria to cultured epithelial cells. The authors also demonstrate that the EPEC-induced flux of IPs precedes actin rearrangement and bacterial invasion. EPEC mutants and tyrosine protein kinase inhibitors were used to establish that formation of IPs is dependent on tyrosine phosphorylation of a 90-kD HeLa protein. Collectively these results suggest that EPEC-induced tyrosine phosphorylation of a host cell substrate(s) leads to release of IPs, which may then trigger cytoskeletal rearrangement.

L7 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 15

TI Signal transduction between enteropathogenic Escherichia coli (EPEC) and epithelial cells: EPEC induces tyrosine phosphorylation of host cell proteins to initiate cytoskeletal rearrangement and bacterial uptake

AB Upon attachment to cultured HeLa cells, enteropathogenic Escherichia coli (EPEC) induces assembly of a complex cytoskeletal structure within the eukaryotic cell, localized beneath the adherent bacterium. In addn., EPEC induces its own internalization by non-phagocytic epithelial cells. It was found that after binding to the epithelial cell surface, EPEC induces tyrosine phosphorylation of three eukaryotic proteins. The major phosphorylation substrate is a 90 kDa protein (***Hp90***). In correlation with ***Hp90*** tyrosine phosphorylation, the EPEC-induced cytoskeletal structure also contained tyrosine phosphorylated proteins. Using tyrosine protein kinase inhibitors and EPEC mutants (cfm) that fail to induce ***Hp90*** phosphorylation, the authors demonstrate that induction of ***Hp90*** phosphorylation is involved in initiation of the cytoskeletal structure assembly and in bacterial uptake. Other non-invasive EPEC mutants (eae) are still able to induce ***Hp90*** tyrosine phosphorylation and to initiate aggregation of the tyrosine phosphorylated proteins and some cytoskeleton components. However, eae mutants are deficient in nucleating the aggregates into an organized structure.

=> s (translocat?)(10n)(intimin)(10n)(receptor?)
L8 90 (TRANSLOCAT?)(10N)(INTIMIN)(10N)(RECEPTOR?)

=> d l10 1-25
L10 ANSWER 1 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 1

AN 2000:472429 CAPLUS

TI Structural basis for recognition of the ***translocated*** ***intimin*** ***receptor*** (Tir) by ***intimin*** from enteropathogenic Escherichia coli

AU Batchelor, Miranda; Prasannan, Sunil; Daniell, Sarah; Reece, Stephen; Connerton, Ian; Bloomberg, Graham; Dougan, Gordon; Frankel, Gad; Matthews, Stephen

CS Department of Biochemistry, Imperial College of Science, Technology and Medicine, London, SW7 2AZ, UK

SO EMBO J. (2000), 19(11), 2452-2464
CODEN: EMJODG; ISSN: 0261-4189

PB Oxford University Press

DT Journal

LA English

L10 ANSWER 2 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 2

AN 2000:51065 CAPLUS

DN 133:16094

TI Antibody response of patients infected with verocytotoxin-producing Escherichia coli to protein antigens encoded on the LEE locus

AU Jenkins, C.; Chart, H.; Smith, H. R.; Hartland, E. L.; Batchelor, M.; Delahay, R. M.; Dougan, G.; Frankel, G.

CS Laboratory of Enteric Pathogens, Central Public Health Laboratory, London, NW9 5HT, UK

SO J. Med. Microbiol. (2000), 49(1), 97-101
CODEN: JMMIAV; ISSN: 0022-2615

PB Lippincott Williams & Wilkins

DT Journal

LA English

RE.CNT 16

RE

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(2) Chart, H; Epidemiol Infect 1998, V120, P239 CAPLUS
(3) Chart, H; Lancet 1998, V352, P371 CAPLUS
(6) Frankel, G; Infect Immun 1994, V62, P1835 CAPLUS
(7) Frankel, G; Mol Microbiol 1998, V30, P911 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS

AN 2000:88974 BIOSIS

DN PREV200000088974

TI Antibody response of patients infected with verocytotoxin-producing Escherichia coli to protein antigens encoded on the LEE locus.

AU Jenkins, C.; Chart, H. (1); Smith, H. R.; Hartland, E. L.; Batchelor, M.; Delahay, R. M.; Dougan, G.; Frankel, G.

CS (1) Laboratory of Enteric Pathogens, Central Public Health Laboratory, 61 Colindale Avenue, London, NW9 5HT UK

SO Journal of Medical Microbiology, (Jan., 2000) Vol. 49, No. 1, pp. 91-101. ISSN: 0022-2615.

DT Article

LA English

SL English

L10 ANSWER 4 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 3

AN 2000:279346 CAPLUS

TI Human colostrum and serum contain antibodies reactive to the ***intimin*** -binding region of the enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor***

AU Sanches, Marcela Imperio; Keller, Rogeria; Hartland, Elizabeth L.; Figueiredo, Dayse M. M.; Batchelor, Miranda; Martinez, Marina B.; Dougan, Gordon; Careiro-Sampaio, Magda M. S.; Frankel, Gad; Trabulsi, Luiz R.

CS Departamento de Microbiologia, Instituto de Ciencias Biomedicas, Departamento de Immunologia, ICB III and Faculdade de Ciencias Farmaceutica, Departamento de Analises Clinicas e Toxicologicas Universidade de Sao Paulo, Sao Paulo, Brazil

SO J. Pediatr. Gastroenterol. Nutr. (2000), 30(1), 73-77
CODEN: JPGND6; ISSN: 0277-2116
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 21
RE
(1) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
(3) Camara, L; Int Arch Allergy Immunol 1994, V103, P307 CAPLUS
(4) Elliott, S; Mol Microbiol 1998, V28, P1 CAPLUS
(5) Frankel, G; Infect Immun 1994, V62, P1835 CAPLUS
(6) Frankel, G; Infect Immun 1996, V64, P5315 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:89908 BIOSIS
DN PREV200000089908
TI Human colostrum and serum contain antibodies reactive to the ***intimin*** -binding region of the enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor*** .
AU Imperio Sanches, Marcela; Keller, Rogeria; Hartland, Elizabeth L.; Figueiredo, Dayse M. M.; Batchelor, Miranda; Martinez, Marina B.; Dougan,
Gordon; Careiro-Sampaio, Magda M. S.; Frankel, Gad (1); Trabulsi, Luiz R.
CS (1) Department of Biochemistry, Imperial College, London, SW7 2AZ UK
SO JPGN, (Jan., 2000) Vol. 30, No. 1, pp. 73-77.
DT Article
LA English
SL English

L10 ANSWER 6 OF 25 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1999-337712 [28] WPIDS
DNN N1999-253081 DNC C1999-099316
TI New ***translocated*** ***intimin*** ***receptor*** useful for
treating infection by enteropathogenic or enterohemorrhagic Escherichia coli.
DC B04 D16 S03
IN DEVINNEY, R; FINLAY, B B; KENNY, B; STEIN, M
PA (UYBR-N) UNIV BRITISH COLUMBIA
CYC 81
PI WO 9924576 A1 19990520 (199928)* EN 91p C12N015-31
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE
GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG
MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 9911373 A 19990531 (199941) C12N015-31
ADT WO 9924576 A1 WO 1998-CA1042 19981110; AU 9911373 A AU 1999-11373 19981110
FDT AU 9911373 A Based on WO 9924576
PRAI US 1997-65130 19971112
IC ICM C12N015-31
ICS A61K038-16; C07K014-24; C07K016-12; C12N015-62; C12Q001-68; G01N033-53

L10 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 4
AN 2000:11301 CAPLUS
DN 132:176532
TI Quorum sensing controls expression of the type III secretion gene transcription and protein secretion in enterohemorrhagic and enteropathogenic Escherichia coli
AU Sperandio, Vanessa; Mellies, Jay L.; Nguyen, William; Shin, Sooan; Kaper,
James B.
CS Center for Vaccine Development and Department of Microbiology and Immunology, University of Maryland School of Medicine, Baltimore, MD, 21201, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1999), 96(26), 15196-15201
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 41
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(8) Elliott, S; Mol Microbiol 1999, V33, P1176 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 25 MEDLINE DUPLICATE 5
AN 1999242825 MEDLINE
DN 99242825
TI Enterohemorrhagic Escherichia coli O157:H7 produces Tir, which is translocated to the host cell membrane but is not tyrosine phosphorylated.
AU DeVinney R; Stein M; Reinscheid D; Abe A; Ruschkowski S; Finlay B B
CS Biotechnology Laboratory, University of British Columbia, Vancouver, British Columbia V6T 1Z3, Canada.
SO INFECTION AND IMMUNITY, (1999 May) 67 (5) 2389-98.
Journal code: GO7. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199907
EW 19990704

L10 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 6
AN 1999:632014 CAPLUS
DN 131:333663
TI Identification of CesT, a chaperone for the type III secretion of Tir in enteropathogenic Escherichia coli
AU Elliott, Simon J.; Hutcheson, Steven W.; Dubois, Maria S.; Mellies, Jay L.; Wainwright, Leslie A.; Batchelor, Miranda; Frankel, Gad; Knutton, Stuart; Kaper, James B.
CS Center for Vaccine Development and Department of Microbiology and Immunology, University of Maryland School of Medicine, Baltimore, MD, 21201, USA
SO Mol. Microbiol. (1999), 33(6), 1176-1189
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 47
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(1) Abe, A; Mol Microbiol 1999, V33 CAPLUS
(2) Anderson, D; Science 1997, V278, P1140 CAPLUS
(5) Cheng, L; Mol Microbiol 1997, V24, P757 CAPLUS
(6) Clark, K; Proc Natl Acad Sci USA 1998, V95, P5401 CAPLUS
(8) Cornelis, G; Microbiol Mol Biol Rev 1998, V62, P1315 CAPLUS
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L10 ANSWER 10 OF 25 MEDLINE DUPLICATE 7
AN 1999440167 MEDLINE
DN 99440167
TI Enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor*** , Tir, requires a specific chaperone for stable secretion.
AU Abe A; de Grado M; Pfuetzner R A; Sanchez-Sanmartin C; Devinney R; Puente
J L; Strynadka N C; Finlay B B
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
Canada V6T 1Z3.
SO MOLECULAR MICROBIOLOGY, (1999 Sep) 33 (6) 1162-75.
Journal code: MOM. ISSN: 0950-382X.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
EW 20000104

L10 ANSWER 11 OF 25 MEDLINE DUPLICATE 8
AN 1999340885 MEDLINE
DN 99340885
TI Enteropathogenic Escherichia coli: a pathogen that inserts its own receptor into host cells.

AU DeVinney R; Gauthier A; Abe A; Finlay B B
CS Biotechnology Laboratory, University of British Columbia, Vancouver, Canada.
SO CELLULAR AND MOLECULAR LIFE SCIENCES, (1999 Jun) 55 (6-7) 961-76. Ref: 122
Journal code: CLE. ISSN: 1420-682X.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals; Cancer Journals
EM 199910
EW 19991002

L10 ANSWER 12 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 9
AN 1999:500094 CAPLUS
DN 131:255689
TI Role of bacterial intimin in colonic hyperplasia and inflammation
AU Higgins, Lisa M.; Frankel, Gad; Connerton, Lan; Goncalves, Nathalie S.; Dougan, Gordon; MacDonald, Thomas T.
CS Department of Paediatric Gastroenterology, St. Bartholomews and the Royal
London School of Medicine and Dentistry, London, EC1A 7BE, UK
SO Science (Washington, D. C.) (1999), 285(5427), 588-591
CODEN: SCIEAS; ISSN: 0036-8075
PB American Association for the Advancement of Science
DT Journal
LA English
RE.CNT 24
RE
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(4) Donnenberg, M; J Bacteriol 1993, V175, P4670 CAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:77113 BIOSIS
DN PREV200000077113
TI Escherichia coli O157:H7 as an emerging foodborne pathogen: A literature review.
AU Park, Sungsu (1); Worobo, Randy W. (1); Durst, Richard A. (1)
CS (1) Department of Food Science and Technology, Cornell University, Geneva,
NY USA
SO Critical Reviews in Food Science and Nutrition, (Nov., 1999) Vol. 39, No.
6, pp. 481-502.
ISSN: 1040-8398.
DT General Review
LA English

L10 ANSWER 14 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:60774 BIOSIS
DN PREV200000060774
TI Interaction of the EPEC protein Tir with focal adhesion proteins.
AU Freeman, Nancy (1); Zurawski, Daniel V. (1); Chowrashi, Prokash (1); Louw,
Andrew (1); Ayoob, Joseph C. (1); Sanger, Joseph W. (1); Sanger, Jean M. (1)
CS (1) University of Pennsylvania, Philadelphia, PA USA
SO Molecular Biology of the Cell, (Nov., 1999) Vol. 10, No. SUPPL., pp. 355a.
Meeting Info.: 39th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 11-15, 1999 The American Society
for Cell Biology
. ISSN: 1059-1524.
DT Conference
LA English

L10 ANSWER 15 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 10
AN 1999:244227 CAPLUS
DN 131:41221
TI Structure of the cell-adhesion fragment of intimin from enteropathogenic Escherichia coli
AU Kelly, Geoff; Prasannan, Sunil; Daniell, Sarah; Fleming, Keiran; Frankel, Gad; Dougan, Gordon; Connerton, Ian; Matthews, Stephen
CS Department of Biochemistry and Centre for Structural Biology, Imperial College of Science, Technology and Medicine, London, SW7 2AY, UK
SO Nat. Struct. Biol. (1999), 6(4), 313-318
CODEN: NSBIEW; ISSN: 1072-8368
PB Nature America
DT Journal
LA English
RE.CNT 46
RE
(1) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
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(6) Delaglio, F; J Biomol NMR 1995, V6, P277 CAPLUS
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L10 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 11
AN 1999:256127 CAPLUS
DN 131:56220
TI Binding of intimin from enteropathogenic Escherichia coli to Tir and to host cells
AU Hartland, Elizabeth L.; Batchelor, Miranda; Delahay, M.; Hale, Christine; Matthews, Stephen; Dougan, Gordon; Knutton, Stuart; Connerton, Ian; Frankel, Gad
CS Department of Biochemistry, Imperial College of Science, Technology and
Medicine, London, SW7 2AZ, UK
SO Mol. Microbiol. (1999), 32(1), 151-158
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 26
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(3) Everest, P; FEMS Microbiol Lett 1995, V126, P97 CAPLUS
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(5) Frankel, G; Infect Immun 1995, V63, P4323 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 17 OF 25 MEDLINE DUPLICATE 12
AN 2000124575 MEDLINE
DN 20124575
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia
coli (EPEC) from dogs and cats.
AU Goffaux F; China B; Janssen L; Pirson V; Mainil J
CS Laboratory of Bacteriology, Faculty of Veterinary Medicine, University of Li`ege, Belgium.
SO ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1999) 473 129-36.
Journal code: 2LU. ISSN: 0065-2598.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200006
EW 20000603

L10 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2000 ACS
AN 2000:444131 CAPLUS
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia
coli (EPEC) from dogs and cats
AU Goffaux, Frederic; China, Bernard; Janssen, Laurence; Pirson, Vinciane; Mainil, Jacques
CS Laboratory of Bacteriology Faculty of Veterinary Medicine, University of Liege, Liege, B-4000, Belg.
SO Adv. Exp. Med. Biol. (1999), 473(Mechanisms in the Pathogenesis of Enteric
Diseases 2), 128-136
CODEN: AEMBAP; ISSN: 0065-2598
PB Kluwer Academic/Plenum Publishers

DT Journal
LA English

L10 ANSWER 19 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 13
AN 1998:711579 CAPLUS
DN 130:92716
TI ***Translocated*** ***intimin*** ***receptors*** (Tir) of Shiga-toxigenic Escherichia coli isolates belonging to serogroups O26, O111, and O157 react with sera from patients with hemolytic-uremic syndrome and exhibit marked sequence heterogeneity
AU Paton, Adrienne; Manning, Paul A.; Woodrow, Matthew C.; Paton, James C.
CS Molecular Microbiology Unit, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Infect. Immun. (1998), 66(11), 5580-5586
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 33
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(1) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
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L10 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 14
AN 1998:297526 CAPLUS
DN 129:51858
TI A novel EspA-associated surface organelle of enteropathogenic Escherichia coli involved in protein translocation into epithelial cells
AU Knutton, Stuart; Rosenshine, Ilan; Pallen, Mark J.; Nisan, Israel; Neves, Bianca C.; Bain, Christopher; Wolff, Carmel; Dougan, Gordon; Frankel, Gad
CS Institute of Child Health, University of Birmingham, Birmingham, B16 8ET,
UK
SO EMBO J. (1998), 17(8), 2166-2176
CODEN: EMJODG; ISSN: 0261-4189
PB Oxford University Press
DT Journal
LA English

L10 ANSWER 21 OF 25 MEDLINE DUPLICATE 15
AN 1999071213 MEDLINE
DN 99071213
TI The medium is the messenger.
AU Phillips A D
CS University Department of Paediatric Gastroenterology, Royal Free Hospital,
Pond Street, London NW3 2QG, UK.
SO GUT, (1998 Oct) 43 (4) 456-7.
Journal code: FVT. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199903
EW 19990301

L10 ANSWER 22 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:373002 BIOSIS
DN PREV199800373002
TI Type III protein secretion systems in bacterial pathogens of animals and plants.
AU Hueck, Christoph J. (1)
CS (1) Biozentrum Univ. Wuerzburg, Am Hubland, 97074 Wuerzburg Germany
SO Microbiology and Molecular Biology Reviews, (June, 1998) Vol. 62, No. 2,
pp. 379-433.
ISSN: 1092-2172.
DT General Review
LA English

L10 ANSWER 23 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 16
AN 1998:588505 CAPLUS
DN 129:299953
TI BipA affects Ca++ fluxes and phosphorylation of the ***translocated*** ***intimin*** ***receptor*** (Tir/Hp90) in host epithelial cells infected with enteropathogenic E. coli
AU Farris, Michele; Grant, Andrew; Jane, Steve; Chad, John; O'Connor, C. David
CS Divisions of Biochemistry and Molecular Biology, School of Biological Sciences, University of Southampton, Southampton, SO16 7PX, UK
SO Biochem. Soc. Trans. (1998), 26(3), S225
CODEN: BCSTB5; ISSN: 0300-5127
PB Portland Press Ltd.
DT Journal
LA English

L10 ANSWER 24 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:415719 BIOSIS
DN PREV199800415719
TI Virulence properties of enteropathogenic Escherichia coli strains of O119:H2 and O128ab:H2 serotypes.
AU Bortolini, M. R. (1); Trabulsi, L. R. (1); Frankel, G.; Sperandio, V. (1)
CS (1) Univ. Sao Paulo, Sao Paulo Brazil
SO Abstracts of the General Meeting of the American Society for Microbiology,
(1998) Vol. 98, pp. 79-80.
Meeting Info.: 98th General Meeting of the American Society for Microbiology Atlanta, Georgia, USA May 17-21, 1998 American Society for Microbiology
. ISSN: 1060-2011.
DT Conference
LA English

L10 ANSWER 25 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 17
AN 1998:777687 CAPLUS
DN 130:107357
TI Molecular and ultrastructural characterization of EspA from different enteropathogenic Escherichia coli serotypes
AU Neves, Bianca C.; Knutton, Stuart; Trabulsi, Luiz R.; Sperandio, Vanessa; Kaper, James B.; Dougan, Gordon; Frankel, Gad
CS Department of Biochemistry, Imperial College of Science, Technology and
Medicine, London, SW7 2AZ, UK
SO FEMS Microbiol. Lett. (1998), 169(1), 73-80
CODEN: FMLED7; ISSN: 0378-1097
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 23
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(1) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l10 1-25 ti,ab
L10 ANSWER 1 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 1
TI Structural basis for recognition of the ***translocated*** ***intimin*** ***receptor*** (Tir) by ***intimin*** from enteropathogenic Escherichia coli
AB Intimin is a bacterial adhesion mol. involved in intimate attachment of enteropathogenic and enterohaemorrhagic Escherichia coli to mammalian host
cells. ***Intimin*** targets the ***translocated*** ***intimin*** ***receptor*** (Tir), which is exported by the bacteria and integrated into the host cell plasma membrane. In this study we localized the Tir-binding region of intimin to the C-terminal 190 amino acids (Int190). We have also detd. the region's high-resoln. soln. structure, which comprises an Ig domain that is intimately coupled to a novel C-type lectin domain. This fragment, which is necessary and sufficient for Tir interaction, defines a new super domain in intimin that exhibits striking structural similarity to the integrin-binding domain of the Yersinia invasin and C-type lectin families. The extracellular

portion of intimin comprises an articulated rod of Ig domains extending from the bacterium surface, conveying a highly accessible "adhesive tip" to the target cell. The interpretation of NMR-titrn. and mutagenesis data has enabled us to identify, for the first time, the binding site for Tir, which is located at the extremity of the Int190 moiety.

L10 ANSWER 2 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 2

TI Antibody response of patients infected with verocytotoxin-producing Escherichia coli to protein antigens encoded on the LEE locus

AB Sera from patients infected with verocytotoxin-producing Escherichia coli (VTEC) O157, from patients with antibodies to E. coli O157 lipopolysaccharide (LPS) and from healthy controls were examd. for antibodies to proteins involved in expressing the attaching and effacing phenotype. After SDS-PAGE, purified recombinant ***intimin*** , EspA-filament structural protein, ***translocated*** protein EspB and three sep. domains of the ***translocated*** ***intimin*** ***receptor*** (Tir) were tested for reaction with patients' sera by immunoblotting. An ELISA was also used to detect antibodies to intimin in sera from E. coli O157 LPS antibody-pos. individuals. Seven of nine culture-pos. patients and one control patient had antibodies to EspA. Five of these patients and two controls had serum antibodies to the intimin-binding region of Tir, whereas none of the sera contained antibodies binding to either of the intracellular domains of Tir. By immunoblotting, 10 of 14 culture-pos. patients had antibodies to the conserved region of intimin, eight of whom were infected with E. coli O157 phage type 2. Thirty six of 60 sera from culture-neg. but E. coli O157 LPS antibody-pos. patients had antibodies to intimin as detd. by ELISA. The secreted proteins are expressed in vivo during infection and are considered as pathogenic markers. Antibodies to these proteins may from the basis of a serodiagnostic test for the detection of patients infected with VTEC which carry the locus for the enterocyte effacement pathogenicity island and provide an adjunct test to the established serol. tests based on VTEC LPS.

L10 ANSWER 3 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS

TI Antibody response of patients infected with verocytotoxin-producing Escherichia coli to protein antigens encoded on the LEE locus.

AB Sera from patients infected with verocytotoxin-producing Escherichia coli (VTEC) O157, from patients with antibodies to E. coli O157 lipopolysaccharide (LPS) and from healthy controls were examined for antibodies to proteins involved in expressing the attaching and effacing phenotype. After SDS-PAGE, purified recombinant ***intimin*** , EspA-filament structural protein, ***translocated*** protein EspB and three separate domains of the ***translocated*** ***intimin*** ***receptor*** (Tir) were tested for reaction with patients' sera by immunoblotting. An ELISA was also used to detect antibodies to intimin in sera from E. coli O157 LPS antibody-positive individuals. Seven of nine culture-positive patients and one control patient had antibodies to EspA. Five of these patients and two controls had serum antibodies to the intimin-binding region of Tir, whereas none of the sera contained antibodies binding to either of the intracellular domains of Tir. By immunoblotting, 10 of 14 culture-positive patients had antibodies to the conserved region of intimin, eight of whom were infected with E. coli O157 phage type 2. Thirty-six of 60 sera from culture-negative but E. coli O157 LPS antibody-positive patients had antibodies to intimin as determined by ELISA. The secreted proteins are expressed in vivo during infection and are considered as pathogenic markers. Antibodies to these proteins may form the basis of a serodiagnostic test for the detection of patients infected with VTEC which carry the locus for the enterocyte effacement pathogenicity island and provide an adjunct test to the established serological tests based on VTEC LPS.

L10 ANSWER 4 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 3

TI Human colostrum and serum contain antibodies reactive to the ***intimin*** -binding region of the enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor***

AB Background: In Brazil, enteropathogenic Escherichia coli (EPEC) diarrhoea is endemic in young infants. A characteristic feature of EPEC adhesion to host cells is intimate attachment leading to the formation of distinctive "attaching and effacing" (A/E) lesions on mammalian cells. Two genes directly involved in intimate adhesion, eae and tir, encode the adhesion mol. ***intimin*** and its ***translocated*** ***receptor*** Tir, resp. The intimin-binding domain of Tir was recently mapped to the middle part of the polypeptide (Tir-M), and the amino (Tir-N) and carboxy (Tir-C) termini were found to be located within infected host cells. Recently, it was shown that colostrum samples from mothers living in Sao Paulo contain IgA-class antibodies reactive with a no. of proteins assocd. with EPEC virulence. It has also been shown that patients infected with verocytotoxin-producing E. coli O157 can produce antibodies to Tir. In the current study antibody responses to the different Tir domains were analyzed in sera and colostrum samples collected in an EPEC-endemic area of Brazil. Methods: Recombinant Tir, Tir-N, Tir-M, and Tir-C were expressed as His-tagged protein in E. coli BL21a and purified on nickel columns. Western blot anal. was used to investigate colostrum IgA- and serum IgG-class antibodies reactive with the Tir fragments. Results: Anti-Tir IgG antibodies were detected in the serum of children, with (63%) or without (50%) diarrhoea. Anti-Tir IgA-class antibodies were detected in all the colostrum pools tested. With the use of both serum IgG- and colostrum IgA-class antibodies, an immunodominant domain of the Tir-polypeptide, Tir M, was identified. Conclusion: The intimin-binding region of Tir (Tir-M) is the immunodominant region of the polypeptide in humans. Both serum IgG-class and colostrum IgA-class antibodies reacted predominantly with the Tir-M domain.

L10 ANSWER 5 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS

TI Human colostrum and serum contain antibodies reactive to the ***intimin*** -binding region of the enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor*** .

AB Background: In Brazil, enteropathogenic Escherichia coli (EPEC) diarrhoea is endemic in young infants. A characteristic feature of EPEC adhesion to host cells is intimate attachment leading to the formation of distinctive "attaching and effacing" (A/E) lesions on mammalian cells. Two genes directly involved in intimate adhesion, eae and tir, encode the adhesion molecule ***intimin*** and its ***translocated*** ***receptor*** Tir, respectively. The ***intimin*** -binding domain of Tir was recently mapped to the middle part of the polypeptide (Tir-M), and the amino (Tir-N) and carboxy (Tir-C) termini were found to be located within infected host cells. Recently, it was shown that colostrum samples from mothers living in Sao Paulo contain IgA-class antibodies reactive with a number of proteins associated with EPEC virulence. It has also been shown that patients infected with verocytotoxin-producing E. coli O157 can produce antibodies to Tir. In the current study antibody responses to the different Tir domains were analyzed in sera and colostrum samples collected in an EPEC-endemic area of Brazil. Methods: Recombinant Tir, Tir-N, Tir-M, and Tir-C were expressed as His-tagged protein in E. coli BL21a and purified on nickel columns. Western blot analysis was used to investigate colostrum IgA- and serum IgG-class antibodies reactive with the Tir fragments. Results: Anti-Tir IgG antibodies were detected in the serum of children, with (63%) or without (50%) diarrhoea. Anti-Tir IgA-class antibodies were detected in all the colostrum pools tested. With the use of both serum IgG- and colostrum IgA-class antibodies, an immunodominant domain of the Tir-polypeptide, Tir M, was identified. Conclusion: The intimin-binding region of Tir (Tir-M) is the immunodominant region of the polypeptide in humans. Both serum IgG-class and colostrum IgA-class antibodies reacted predominantly with the Tir-M domain.

L10 ANSWER 6 OF 25 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD

TI New ***translocated*** ***intimin*** ***receptor*** useful for treating infection by enteropathogenic or enterohemorrhagic Escherichia coli.

AB WO 9924576 A UPAB: 19990719

NOVELTY - A ***translocated*** ***intimin*** ***receptor*** (Tir) polypeptide that binds ***intimin*** , is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (I) encoding Tir;

(2) a polynucleotide selected from:

(a) the 1920 bp sequence (Ia) given in the specification;

(b) (Ia) where T is U;

(c) nucleic acid sequences complementary to (a) or (b);

(d) fragments of (a), (b) or (c) that are at least 15 nucleotides long and that hybridize to DNA which encode the 549 amino acid polypeptide defined in the specification;

(3) a polynucleotide selected from:

(a) the 1723 bp sequence (Ib) given in the specification;

(b) (Ib) where T is U;

(c) nucleic acid sequences complementary to (a) or (b);

(d) fragments of (a), (b) or (c) that are at least 15 nucleotides

long and that hybridize to DNA which encode the 559 amino acid polypeptide
defined in the specification;
(4) a vector containing (I);
(5) a host cell containing the vector of (4);
(6) an anti-Tir antibody;
(7) detecting Tir or its polynucleotides in a sample comprising:
(a) contacting the sample with an anti-Tir antibody or a nucleic acid probe that hybridizes to the Tir polynucleotide;
(b) detecting binding of the antibody to Tir polypeptide, where binding is indicative of the presence of the Tir polypeptide in the sample; or hybridization of the probe with the Tir polynucleotide which is indicative of Tir polynucleotide in the sample;
(8) a recombinant method for the production of Tir polynucleotides and polypeptides;
(9) a polynucleotide produced by (8);
(10) a host cell containing the polynucleotide of (9);
(11) production of a Tir fusion protein;
(12) identifying a compound that interferes with binding of Tir to intimin comprises comparing the binding of the Tir polypeptide to intimin in the presence and absence of the compound ;
(13) a method for differentiating among attaching and effacing pathogens by contacting them with an anti-Tir antibody and an anti-phosphotyrosine antibody;
(14) delivering a compound of interest to a Tir-containing cell by administering to the cell an intimin-containing cell delivery vehicle that contains a compound of interest;
(15) kits for detection of Tir polypeptides or polynucleotides; and
(16) a method for inducing a cell-mediated immune response to a polypeptide of interest, by contacting a subject with an attenuated bacteria, where the bacteria lacks an EspA or EspB protein and where the bacteria contains a polynucleotide encoding a Tir fusion protein.
ACTIVITY - Antibacterial.
MECHANISM OF ACTION - Vaccine.
USE - Tir antibodies can be used to detect Tir in tissue or biological fluids, where presence of Tir is indicative of infection by enteropathogenic or enterohemorrhagic Escherichia coli (designated EPEC and EHEC, respectively). The antibody is able to differentiate among attaching and effacing pathogens, when used in conjunction with an anti-phosphotyrosine antibody. Tir can be used to induce an immune response in humans or cows against EPEC or EHEC to ameliorate diseases caused by the Tir-producing EPEC or EHEC. Tir polynucleotides can be used
as probes to detect the presence of Tir polynucleotides in a sample. Tir can also be used to detect a cell cytoskeleton. Additionally, Tir can be used to identify compounds that interfere with Tir binding to intimin. The Tir fusion proteins can be used in attenuated Escherichia coli to induce a cell-mediated immune response to polypeptides of interest, e.g. antigens (all Claimed).
Dwg.0/9

L10 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 4
TI Quorum sensing controls expression of the type III secretion gene transcription and protein secretion in enterohemorrhagic and enteropathogenic Escherichia coli
AB Enterohemorrhagic Escherichia coli O157:H7 and enteropathogenic E. coli
cause a characteristic histopathol. in intestinal cells known as attaching and effacing. The attaching and effacing lesion is encoded by the Locus of Enterocyte Effacement (LEE) pathogenicity island, which encodes a type III secretion system, the ***intimin*** intestinal colonization factor, and the ***translocated*** ***intimin*** ***receptor*** protein that is ***translocated*** from the bacterium to the host epithelial cells. Using lacZ reporter gene fusions, the authors show that expression of the LEE operons encoding the type III secretion system, ***translocated*** ***intimin*** ***receptor*** , and ***intimin*** is regulated by quorum sensing in both enterohemorrhagic E. coli and enteropathogenic E. coli. The luxS gene recently shown to be responsible for prodn. of autoinducer in the Vibrio harveyi and E. coli quorum-sensing systems is responsible for regulation of the LEE operons, as shown by the mutation and complementation of the luxS gene. Regulation
of intestinal colonization factors by quorum sensing could play an important role in the pathogenesis of disease caused by these organisms. These results suggest that intestinal colonization by E. coli O157:H7, which has an unusually low infectious dose, could be induced by quorum sensing of signals produced by nonpathogenic E. coli of the normal intestinal flora.

L10 ANSWER 8 OF 25 MEDLINE DUPLICATE 5
TI Enterohemorrhagic Escherichia coli O157:H7 produces Tir, which is translocated to the host cell membrane but is not tyrosine phosphorylated.
AB Intimate attachment to the host cell leading to the formation of attaching and effacing (A/E) lesions is an essential feature of enterohemorrhagic Escherichia coli (EHEC) O157:H7 pathogenesis. In a related pathogen, enteropathogenic E. coli (EPEC), this activity is dependent upon ***translocation*** of the ***intimin*** ***receptor*** , Tir, which becomes tyrosine phosphorylated within the host cell membrane. In contrast, the accumulation of tyrosine-phosphorylated proteins beneath adherent EHEC bacteria does not occur, leading to questions about whether EHEC uses a Tir-based mechanism for adherence and A/E lesion formation. In
this report, we demonstrate that EHEC produces a functional Tir that is inserted into host cell membranes, where it serves as an intimin receptor. However, unlike in EPEC, in EHEC Tir is not tyrosine phosphorylated yet plays a key role in both bacterial adherence to epithelial cells and pedestal formation. EHEC, but not EPEC, was unable to synthesize Tir in Luria-Bertani medium but was able to secrete Tir into M9 medium, suggesting that Tir synthesis and secretion may be regulated differently in these two pathogens. EHEC Tir and EPEC Tir both bind intimin and focus
cytoskeletal rearrangements, indicating that tyrosine phosphorylation is not needed for pedestal formation. EHEC and EPEC intimins are functionally
interchangeable, but EHEC Tir shows a much greater affinity for EHEC intimin than for EPEC intimin. These findings highlight some of the differences and similarities between EHEC and EPEC virulence mechanisms,
which can be exploited to further define the molecular basis of pedestal formation.

L10 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 6
TI Identification of CesT, a chaperone for the type III secretion of Tir in enteropathogenic Escherichia coli
AB The locus of enterocyte effacement of enteropathogenic Escherichia coli encodes a type III secretion system, an outer membrane protein adhesin (***intimin*** , the product of eae) and Tir, a ***translocated*** protein that becomes a host cell ***receptor*** for ***intimin*** . Many type III secreted proteins require chaperones, which function to stabilize proteins, prevent inappropriate protein-protein interactions and aid in secretion. An open reading frame located between tir and eae, previously named orfU, was predicted to encode a protein with partial similarity to the Yersinia SycH chaperone. The authors examd. the potential of the orfU gene product to serve as a chaperone for Tir. The orfU gene encoded a 15 kDa cytoplasmic protein that specifically interacted with Tir as demonstrated by the yeast two-hybrid assay, column binding and coimmunopptn. expts. An orfU mutant was defective in attaching-effacing lesion formation and Tir secretion, but was unaffected in expression of other virulence factors. OrfU appeared to stabilize Tir levels in the cytoplasm, but was not absolutely necessary for secretion of Tir. Based upon the phys. similarities, phenotypic characteristics and the demonstrated interaction with Tir, orfU is redesignated as cesT for the chaperone for E. coli secretion of Tir.

L10 ANSWER 10 OF 25 MEDLINE DUPLICATE 7
TI Enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor*** , Tir, requires a specific chaperone for stable secretion.
AB Enteropathogenic Escherichia coli (EPEC) secretes several Esps (E. coli-secreted proteins) that are required for full virulence. Insertion of the bacterial protein Tir into the host epithelial cell membrane is facilitated by a type III secretion apparatus, and at least EspA and EspB are required for Tir translocation. An EPEC outer membrane protein, intimin, interacts with Tir on the host membrane to establish intimate attachment and formation of a pedestal-like structure. In this study, we identified a Tir chaperone, CesT, whose gene is located between tir and eae (which encodes intimin). A mutation in cesT abolished Tir secretion into culture supernatants and significantly decreased the amount of Tir in the bacterial cytoplasm. In contrast, this mutation did not affect the secretion of the Esp proteins. The level of tir mRNA was not affected by the cesT mutation, indicating that CesT acts at the post-transcriptional level. The cesT mutant could not induce host cytoskeletal rearrangements, and displayed the same phenotype as the tir mutant. Gel overlay and GST pulldown assays demonstrated that CesT specifically interacts with Tir, but not with other Esp proteins. Furthermore, by using a series of Tir deletion derivatives, we determined that the CesT binding domain is located within the first 100 amino-terminal residues of Tir, and that the

pool of Tir in the bacterial cytoplasm was greatly reduced when this domain was disrupted. Interestingly, this domain was not sufficient for Tir secretion, and at least the first 200 residues of Tir were required for efficient secretion. Gel filtration studies showed that Tir-CesT forms a large multimeric complex. Collectively, these results indicate that CesT is a Tir chaperone that may act as an anti-degradation factor by specifically binding to its amino-terminus, forming a multimeric stabilized complex.

L10 ANSWER 11 OF 25 MEDLINE DUPLICATE 8
TI Enteropathogenic Escherichia coli: a pathogen that inserts its own receptor into host cells.
AB Enteropathogenic Escherichia coli (EPEC) is a major cause of infant diarrhea, killing hundreds of thousands of children per year worldwide. Intimate attachment to the host cell leading to the formation of actin-rich pedestals beneath the adhering bacteria is an essential feature of EPEC pathogenesis. EPEC attaches to host cells via the outer membrane adhesin, intimin. It was recently shown that EPEC inserts its own ***receptor*** for intimate adherence, Tir (***translocated*** ***intimin*** ***receptor***) into the host cell membrane. The focus of this review is on the discovery and characterization of this novel receptor, and our current understanding of its role in pedestal formation. Gram-negative bacterial secretion systems, including type III secretion systems, are reviewed and discussed in the context of Tir delivery into the host cell membrane. The relationship and relevance of in vitro models compared to the actual in vivo situation is essential to understanding disease. We have critically reviewed the use of animal models in studying EPEC infection. Elucidating the function of Tir will contribute to our understanding of how EPEC mediates disease.

L10 ANSWER 12 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 9
TI Role of bacterial intimin in colonic hyperplasia and inflammation
AB Enteropathogenic Escherichia coil (EPEC) cells adhere to gut epithelial cells through intimin a: the Ligand for a bacterially derived epithelial transmembrane protein called the ***translocated*** ***intimin*** ***receptor*** . Citrobacter rodentium colonizes the mouse colon in a similar fashion and uses a different intimin: intimin .beta.. Intimin .alpha. was found to costimulate submitogenic signals through the T cell receptor. Dead intimin .beta.+ C. rodentium, intimin .alpha.-transfected C. rodentium or E. coli strain K12, and EPEC induced mucosal hyperplasia identical to that caused by C. rodentium live infection, as well as a massive T helper cell-type 1 immune response in the colonic mucosa. Mutation of cysteine-937 of intimin to alanine reduced costimulatory activity in vitro and prevented immunopathol. in vivo. The mucosal changes elicited by C. rodentium were interferon-.gamma.-dependent. Immunopathol. induced by intimin enables the bacteria to promote conditions that are favorable for increased microbial colonization.

L10 ANSWER 13 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
TI Escherichia coli O157:H7 as an emerging foodborne pathogen: A literature review.

L10 ANSWER 14 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
TI Interaction of the EPEC protein Tir with focal adhesion proteins.

L10 ANSWER 15 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 10
TI Structure of the cell-adhesion fragment of intimin from enteropathogenic Escherichia coli
AB Enteropathogenic Escherichia coli (EPEC) induce gross cytoskeletal rearrangement within epithelial cells, immediately beneath the attached bacterium. The C-terminal 280 amino acid residues of intimin (Int280; 30.1 kDa), a bacterial cell-adhesion mol., mediate the intimate bacterial host-cell interaction. Recently, interest in this process has been stimulated by the discovery that the bacterial ***intimin*** ***receptor*** protein (Tir) is ***translocated*** into the host cell membrane, phosphorylated, and after binding ***intimin*** triggers the intimate attachment. Using multidimensional NMR and combining perdeuteration with site-specific protonation of Me groups, we have detd. the global fold of Int280. This represents one of the largest, non-oligomeric protein structures to be detd. by NMR that has not been previously resolved by X-ray crystallog. Int280 comprises three domains; two Ig-like domains and a C-type lectin-like module, which define a new family of bacterial adhesion mols. These findings also imply that carbohydrate recognition may be important in intimin-mediated cell adhesion.

L10 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 11
TI Binding of intimin from enteropathogenic Escherichia coli to Tir and to host cells
AB Enteropathogenic Escherichia coli (EPEC) induce characteristic attaching and effacing (A/E) lesions on epithelial cells. This event is mediated, in part, by binding of the bacterial outer membrane protein, ***intimin*** , to a second EPEC protein, Tir (***translocated*** ***intimin*** ***receptor***), which is exported by the bacteria and integrated into the host cell plasma membrane. In this study, we have localized the intimin-binding domain of Tir to a central 107-amino-acid region, designated Tir-M. We provide evidence that both the amino- and carboxy-termini of Tir are located within the host cell. In addn., using immunogold labeling electron microscopy, we have confirmed that intimin can bind independently to host cells even in the absence of Tir. This Tir-independent interaction and the ability of EPEC to induce A/E lesions requires an intact lectin-like module residing at the carboxy-terminus of the intimin polypeptide. Using the yeast two-hybrid system and gel overlays, we show that intimin can bind both Tir and Tir-M even when the lectin-like domain is disrupted. These data provide strong evidence that intimin interacts not only with Tir but also in a lectin-like manner with a host cell intimin receptor.

L10 ANSWER 17 OF 25 MEDLINE DUPLICATE 12
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia coli (EPEC) from dogs and cats.
AB Enteropathogenic Escherichia coli (EPEC) produce attaching and effacing lesions. The genes responsible for this lesion are clustered on the chromosome forming a 35.5 kilobase pathogenesis island called LEE. The LEE was identified, characterized and completely sequenced from the human EPEC strain E2348/69. The LEE carries genes coding for: a type III secretion system (genes esc and sep), the ***translocated*** ***intimin*** ***receptor*** (gene tir), the outer membrane protein ***intimin*** (gene eae) and the E. coli secreted proteins EspA, EspB, and EspD (genes esp). In addition to man and farm animals, EPEC are also isolated from dogs and cats. We studied structurally and functionally the LEE of dog and cat EPEC. First, we used four probes scattered along the LEE to identify the presence of a LEE in canine and feline EPEC isolates. Second, by PCR, we checked the presence of genes homologous to eae, sep, esp, and tir genes in these strains. Third, since the four types of eae and tir genes were described, we developed a multiplex PCR in order to determine the type of eae and tir genes present in each strain. Fourth, we determined by PCR the site of the LEE insertion on the chromosome. Fifth, we tested several of the canine EPEC in their capacity to induce attaching and effacing lesions in the rabbit intestinal loop assay. We can conclude from this study: first, that the a LEE-like structure is present in all tested strains and that it contains genes homologous to esp, sep, tir, and eae genes; second, that there is some preferential associations between the type of eae gene and the type of tir gene present in a strain; third, that the majority of the tested strains contained a LEE located elsewhere on the chromosome in comparison to the human EPEC strain E2348/69; and fourth that dog EPEC were able to induce attaching and effacing lesions in rabbit ileal loop assay.

L10 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2000 ACS
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia coli (EPEC) from dogs and cats
AB Enteropathogenic Escherichia coli (EPEC) produce attaching and effacing lesions. The genes responsible for this lesion are clustered on the chromosome forming a 35.5 kilobase pathogenesis island called LEE. The LEE was identified, characterized and completely sequenced from the human EPEC strain E2348/69. The LEE carries genes coding for: a type III secretion system (genes esc and sep), the ***translocated*** ***intimin*** ***receptor*** (gene tir), the outer membrane protein ***intimin*** (gene eae) and the E. coli secreted proteins EspA, EspB, and EspD (genes esp). In addn. to man and farm animals, EPEC are also isolated from dogs and cats. We studied structurally and functionally the LEE of dog and cat EPEC. First, we used four probes scattered along the LEE to identify the presence of a LEE in canine and feline EPEC isolates. Second, by PCR, we checked the presence of genes homologous to eae, sep, esp, and tir genes in these strains. Third, since the four types of eae and tir genes were described, we developed a multiplex PCR in order to det. the type of eae and tir genes present in each strain. Fourth, we

detd. by PCR the site of the LEE insertion on the chromosome. Fifth, we tested several of the canine EPEC in their capacity to induce attaching and effacing lesions in the rabbit intestinal loop assay. We can conclude from this study: first, that the a LEE-like structure is present in all tested strains and that it contains genes homologous to esp, sep, tir, and eae genes; second, that there is some preferential assocns. between the type of eae gene and the type of tir gene present in a strain; third, that the majority of the tested strains contained a LEE located elsewhere on the chromosome in comparison to the human EPEC strain E2348/69; and fourth
that dog EPEC were able to induce attaching and effacing lesions in rabbit ileal loop assay.

L10 ANSWER 19 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 13
TI ***Translocated*** ***intimin*** ***receptors*** (Tir) of Shiga-toxigenic Escherichia coli isolates belonging to serogroups O26, O111, and O157 react with sera from patients with hemolytic-uremic syndrome and exhibit marked sequence heterogeneity
AB The capacity to form attaching and effacing (A/E) lesions on the surfaces of enterocytes is an important virulence trait of several enteric pathogens, including enteropathogenic Escherichia coli (EPEC) and Shiga-toxigenic E. coli (STEC). Formation of such lesions depends upon an interaction between a bacterial outer membrane protein (intimin) and a bacterially encoded receptor protein (Tir) which is exported from the bacterium and translocated into the host cell membrane. Intimin, Tir, and several other proteins necessary for generation of A/E lesions are encoded on a chromosomal pathogenicity island termed the locus for enterocyte effacement (LEE). Reports of sequence heterogeneity and antigenic variation in the region of intimin believed to be responsible for receptor binding raise the possibility that the receptor itself is also heterogeneous. We have examd. this by cloning and sequencing tir genes from three different STEC strains belonging to serogroups O26, O111, and O157. The deduced amino acid sequences for the Tir homologs from these strains varied markedly, exhibiting only 65.4, 80.2, and 56.7% identity, resp., to that recently reported for EPEC Tir. STEC Tir is also highly immunogenic in humans. Western blots of E. coli DH5.alpha. expressing the
various STEC tir genes cloned in pBluescript [but not E. coli DH5.alpha.(pBluescript)] reacted strongly with convalescent sera from patients with hemolytic-uremic syndrome (HUS) caused by known LEE-pos.
STEC. Moreover, no reaction was seen when the various clone lysates were probed with serum from a patient with HUS caused by a LEE-neg. STEC or with serum from a healthy individual. Covariation of exposed epitopes on both intimin and Tir may be a means whereby STEC avoid host immune responses without compromising adhesin-receptor interaction.

L10 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 14
TI A novel EspA-associated surface organelle of enteropathogenic Escherichia coli involved in protein translocation into epithelial cells
AB Enteropathogenic Escherichia coli (EPEC), like many bacterial pathogens, employ a type III secretion system to deliver effector proteins across the bacterial cell. In EPEC, four proteins are known to be exported by a type III secretion system-EspA, EspB and EspD required for subversion of host cell signal transduction pathways and a ***translocated*** ***intimin*** ***receptor*** (Tir) protein (formerly Hp90) which is tyrosine-phosphorylated following transfer to the host cell to become a receptor for intimin-mediated intimate attachment and "attaching and effacing" (A/E) lesion formation. The structural basis for protein translocation has yet to be fully elucidated for any type III secretion system. A novel EspA-contg. filamentous organelle is described that is present on the bacterial surface during the early stage of A/E lesion formation, forms a phys. bridge between the bacterium and the infected eukaryotic cell surface and is required for the translocation of EspB into infected epithelial cells.

L10 ANSWER 21 OF 25 MEDLINE DUPLICATE 15
TI The medium is the messenger.
AB Enteropathogenic Escherichia coli (EPEC), like many bacterial pathogens, employ a type III secretion system to deliver effector proteins across the bacterial cell. In EPEC, four proteins are known to be exported by a type III secretion system--EspA, EspB and EspD required for subversion of host cell signal transduction pathways and a ***translocated*** ***intimin*** ***receptor*** (Tir) protein (formerly Hp90) which is tyrosine-phosphorylated following transfer to the host cell to become a receptor for intimin-mediated intimate attachment and "attaching and effacing" (A/E) lesion formation. The structural basis for protein translocation has yet to be fully elucidated for any type III secretion system. Here, we describe a novel EspA-containing filamentous organelle that is present on the bacterial surface during the early stage of A/E lesion formation, forms a physical bridge between the bacterium and the infected eukaryotic cell surface and is required for the translocation of EspB into infected epithelial cells.

L10 ANSWER 22 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
TI Type III protein secretion systems in bacterial pathogens of animals and plants.

L10 ANSWER 23 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 16
TI BipA affects Ca++ fluxes and phosphorylation of the ***translocated*** ***intimin*** ***receptor*** (Tir/Hp90) in host epithelial cells infected with enteropathogenic E. coli
AB The capacity of enteropathogenic Escherichia coli (EPEC) to cause diarrheal disease is closely linked to their ability to adhere intimately to the epithelial cells of the small intestine and to trigger complex cytoskeletal rearrangements, result in the attachment and effacing (AE) lesions. Intimate attachment of the bacteria requires interaction of an EPEC outer membrane protein with a tyrosine phosphorylated receptor, Tir/Hp90, located in the epithelial cell membrane. This receptor is an EPEC-encoded protein that is translocated into the host cell where it becomes phosphorylated and assembles underneath the bacteria. BipA, a novel GTPase, has been shown to regulate EPEC processes, including the ability of the bacteria to trigger cytoskeletal rearrangements which result in AC lesions. The effects of BipA on Ca2+ reflux and Tir phosphorylation were tested in Hela cells infected with wild-type, null, or BipA-overproducing EPEC. The results showed that the degree of Ca2+ trigger by BipA does not reflect the severity of cytoskeletal rearrangements. BipA GTPase influences either the prodn. of Tir or its tyrosine phosphorylation in host cells, but Tir may not required for the formation of attaching and effacing lesions.

L10 ANSWER 24 OF 25 BIOSIS COPYRIGHT 2000 BIOSIS
TI Virulence properties of enteropathogenic Escherichia coli strains of O119:H2 and O128ab:H2 serotypes.

L10 ANSWER 25 OF 25 CAPLUS COPYRIGHT 2000 ACS
DUPLICATE 17
TI Molecular and ultrastructural characterization of EspA from different enteropathogenic Escherichia coli serotypes
AB Enteropathogenic Escherichia coli (EPEC) encode a type III secretion system located on a pathogenicity island known as the locus for enterocyte effacement. Four proteins are known to be exported by this type II secretion system - EspA, EspB and EspD required for subversion of host cell signal transduction pathways and a ***translocated*** ***intimin*** ***receptor*** protein (Tir) required for ***intimin*** -mediated intimate attachment and attaching and effacing lesion formation. The espA gene is located within the locus for enterocyte effacement and the EspA polypeptide from the prototype EPEC strain E2348/69 (0127:H6) has recently been shown to be a component of a filamentous structure involved in bacteria-host cell interaction and locus for enterocyte effacement-encoded protein translocation involved in attaching and effacing lesion formation. In this study the authors have extended our investigation of EspA to strains belonging to other classical EPEC serotypes. DNA sequencing demonstrated that the espA gene from the
different EPEC strains share at least 65% DNA identity. In addn., the authors detected morphol. and antigenically similar EspA filaments in all but one of the bacterial strains examd. including recombinant, non-pathogenic E. coli expressing espA from a cloned locus for enterocyte effacement region (HB101(pCVD462)).

=> s l10 and (sequence?)
L11 8 L10 AND (SEQUENCE?)

=> d l11 1-8
L11 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 2000:444131 CAPLUS
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia
coli (EPEC) from dogs and cats
AU Goffaux, Frederic; China, Bernard; Janssen, Laurence; Pirson, Vinciane; Mainil, Jacques
CS Laboratory of Bacteriology Faculty of Veterinary Medicine, University of Liege, Liege, B-4000, Belg.

SO Adv. Exp. Med. Biol. (1999), 473(Mechanisms in the Pathogenesis of Enteric
Diseases 2), 128-136
CODEN: AEMBAP; ISSN: 0065-2598
PB Kluwer Academic/Plenum Publishers
DT Journal
LA English

L11 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1999:632014 CAPLUS
DN 131:333663
TI Identification of CesT, a chaperone for the type III secretion of Tir in enteropathogenic Escherichia coli
AU Elliott, Simon J.; Hutcheson, Steven W.; Dubois, Maria S.; Mellies, Jay L.; Wainwright, Leslie A.; Batchelor, Miranda; Frankel, Gad; Knutton, Stuart; Kaper, James B.
CS Center for Vaccine Development and Department of Microbiology and Immunology, University of Maryland School of Medicine, Baltimore, MD, 21201, USA
SO Mol. Microbiol. (1999), 33(6), 1176-1189
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 47
RE
(1) Abe, A; Mol Microbiol 1999, V33 CAPLUS
(2) Anderson, D; Science 1997, V278, P1140 CAPLUS
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(8) Cornelis, G; Microbiol Mol Biol Rev 1998, V62, P1315 CAPLUS
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L11 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1998:777687 CAPLUS
DN 130:107357
TI Molecular and ultrastructural characterization of EspA from different enteropathogenic Escherichia coli serotypes
AU Neves, Bianca C.; Knutton, Stuart; Trabulsi, Luiz R.; Sperandio, Vanessa; Kaper, James B.; Dougan, Gordon; Frankel, Gad
CS Department of Biochemistry, Imperial College of Science, Technology and
Medicine, London, SW7 2AZ, UK
SO FEMS Microbiol. Lett. (1998), 169(1), 73-80
CODEN: FMLED7; ISSN: 0378-1097
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 23
RE
(1) Adu-Bobie, J; J Clin Microbiol 1998, V36, P662 CAPLUS
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(4) Frankel, G; Infect Immun 1994, V62, P1835 CAPLUS
(5) Gomez-Duarte, O; Infect Immun 1995, V63, P1767 CAPLUS
(6) Jarvis, K; Proc Natl Acad Sci USA 1995, V92, P7996 CAPLUS
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L11 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1998:711579 CAPLUS
DN 130:92716
TI ***Translocated*** ***intimin*** ***receptors*** (Tir) of Shiga-toxigenic Escherichia coli isolates belonging to serogroups O26, O111, and O157 react with sera from patients with hemolytic-uremic syndrome and exhibit marked ***sequence*** heterogeneity
AU Paton, Adrienne; Manning, Paul A.; Woodrow, Matthew C.; Paton, James C.
CS Molecular Microbiology Unit, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Infect. Immun. (1998), 66(11), 5580-5586
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 33
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(2) Altschul, S; J Mol Biol 1990, V215, P403 CAPLUS
(3) Bairoch, A; Nucleic Acids Res 1992, V20, P2013 CAPLUS
(5) Beebakhee, G; FEMS Microbiol Lett 1992, V91, P63 CAPLUS
(7) Butterton, J; Infect Immun 1997, V65, P2127 CAPLUS
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L11 ANSWER 5 OF 8 MEDLINE
AN 2000124575 MEDLINE
DN 20124575
TI The locus for enterocyte effacement (LEE) of enteropathogenic Escherichia
coli (EPEC) from dogs and cats.
AU Goffaux F; China B; Janssen L; Pirson V; Mainil J
CS Laboratory of Bacteriology, Faculty of Veterinary Medicine, University of Li`ege, Belgium.
SO ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1999) 473 129-36.
Journal code: 2LU. ISSN: 0065-2598.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200006
EW 20000603

L11 ANSWER 6 OF 8 MEDLINE
AN 1999440167 MEDLINE
DN 99440167
TI Enteropathogenic Escherichia coli ***translocated*** ***intimin*** ***receptor*** , Tir, requires a specific chaperone for stable secretion.
AU Abe A; de Grado M; Pfuetzner R A; Sanchez-Sanmartin C; Devinney R; Puente
J L; Strynadka N C; Finlay B B
CS Biotechnology Laboratory, University of British Columbia, Vancouver, BC,
Canada V6T 1Z3.
SO MOLECULAR MICROBIOLOGY, (1999 Sep) 33 (6) 1162-75.
Journal code: MOM. ISSN: 0950-382X.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
EW 20000104

L11 ANSWER 7 OF 8 MEDLINE
AN 1999242825 MEDLINE
DN 99242825
TI Enterohemorrhagic Escherichia coli O157:H7 produces Tir, which is translocated to the host cell membrane but is not tyrosine phosphorylated.
AU DeVinney R; Stein M; Reinscheid D; Abe A; Ruschkowski S; Finlay B B
CS Biotechnology Laboratory, University of British Columbia, Vancouver, British Columbia V6T 1Z3, Canada.
SO INFECTION AND IMMUNITY, (1999 May) 67 (5) 2389-98.
Journal code: GO7. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199907
EW 19990704

L11 ANSWER 8 OF 8 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1999-337712 [28] WPIDS
DNN N1999-253081 DNC C1999-099316
TI New ***translocated*** ***intimin*** ***receptor*** useful for
treating infection by enteropathogenic or enterohemorrhagic Escherichia coli.
DC B04 D16 S03
IN DEVINNEY, R; FINLAY, B B; KENNY, B; STEIN, M
PA (UYBR-N) UNIV BRITISH COLUMBIA
CYC 81
PI WO 9924576 A1 19990520 (199928)* EN 91p C12N015-31
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE
GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG

MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 9911373 A 19990531 (199941) C12N015-31
ADT WO 9924576 A1 WO 1998-CA1042 19981110; AU 9911373 A AU 1999-11373 19981110
FDT AU 9911373 A Based on WO 9924576
PRAI US 1997-65130 19971112
IC ICM C12N015-31
ICS A61K038-16; C07K014-24; C07K016-12; C12N015-62; C12Q001-68; G01N033-53

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(FILE 'HOME' ENTERED AT 18:51:31 ON 17 JUL 2000)

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, SCISEARCH, WPIDS' ENTERED AT
18:51:36 ON 17 JUL 2000
E FINLAY B/AU
L1 1288 S E3-E16
E KENNY B/AU
L2 262 S E3,E13,E14,E6
E DE VINNEY/AU
L3 5 S E4,E1
E DEVINNEY/AU
L4 63 S E1,E2,E14-E17
E DEVINNY R/AU
E VINNY R/AU
E VINNEY R/AU
E STEIN M/AU
L5 2020 S E3,E41
L6 69 S (L1 OR L2 OR L3 OR L4 OR L5) AND (INTIMIN OR HP90 OR HP 9
L7 21 DUPLICATE REMOVE L6 (48 DUPLICATES REMOVED)
L8 90 S (TRANSLOCAT?)(10N)(INTIMIN)(10N)(RECEPTOR?)
L9 80 S L8 NOT L7
L10 25 DUPLICATE REMOVE L9 (55 DUPLICATES REMOVED)
L11 8 S L10 AND (SEQUENCE?)